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THE STOKES-ADAMS SYNDROME: ITS RELATIONSHIP TO AURICULO-VENTRICULAR DISSOCIATION AND OTHER ALLIED CONDITIONS.

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HISTORICAL SURVEY.

THE association of cerebral symptoms with an abnormal slowness of the pulse rate was first described by Morgagni[1] who in 1773 described a case of "epilepsy with a slow pulse," and a similar case was recorded by Spens in 1792. An historic example is that of Napoleon I who suffered from "epileptic" attacks in his youth; his pulse during life was persistently slow, which fact was considered by Corvisart as *"une marque de son sang froid et du parfait équilibre de sa volonté."*

It was not until 1826 that a correlation of these two conditions was attempted. Adams,[2] a Dublin physician, published a full account of the case of a man aged sixty-eight, who during the last seven years of his life had suffered from some twenty attacks of apoplexy, none of which was followed by paralysis. The pulse rate was at all times remarkably slow, about 30, and during these attacks it was even slower. From the *post mortem* evidence which showed gross fatty infiltration of the heart and interventricular septum, Adams concluded that "the apoplexy must be considered less a disease in itself than symptomatic of one, the organic seat of which was in the heart."

Stokes,[3] also of Dublin, some twenty years later recorded another similar case in which, in addition to "apoplectic" attacks and a slow pulse, he noted that the venous pulse in the neck was more than twice that of the radial pulse. He was at a loss to explain this and stated that "the pseudo-apoplectic attacks were attributable to an enfeebled circulation . . . . and presented a combination of cerebral and cardiac phenomena of which our knowledge is still imperfect."

Attention was soon diverted from the cardiogenic origin of such cases, as suggested by Adams and Stokes, by the experimental work of the brothers Weber in 1846. They showed that in animals stimulation of the peripheral cut end of the vagus nerve slowed the heart beat and from this arose the neurogenic theory and for nearly fifty years it held sway. Charcot in 1877 expressed the current opinion in stating that "certain slow pulses were the result of medical or surgical lesions of the medulla and spinal cord." This was universally accepted until the end of the last century.

The modern conception may be considered to have its origin in the experimental work of Stannius.[4] His second ligature showed that the ventricle has the faculty of initiating its own beat when dissociated from the auricle, but his conclusions were based upon an erroneous theory of propagation of the heart beat. His in 1893 showed that the impulse to ventricular contraction was passed on from the auricle by means of a neuro-muscular bundle, which now bears his name, situated in the region of the septum. He further showed that interruption of this bundle blocked the passage of such impulse and produced various incoordinations of auricular and ventricular contraction. With complete interruption of the bundle a dissociation occurred between auricle and ventricle, each beating at its own rate. His work was confirmed by Gaskell.

Clinical confirmation of this work was first given in a case by Handford in 1904[5] in which gummata of the heart caused death from heart block. Further cases were soon reported.[6] Mackenzie in 1905[7] showed that conduction in the bundle was influenced by some drugs, particularly digitalis.

Erlanger[8] in 1906 confirmed the relationship between clinical phenomena and the experimental work and produced syncopal attacks in animals by disturbance of ventricular rate. He further showed that vagal slowing of the ventricle did not occur if the bundle was completely destroyed, whilst the accelerator mechanism remained unaffected.

Frédéricq[9] has more recently shown that in certain cases of complete heart block the ventricle in experiment is susceptible to vagal influence and has sought to explain this by a theory of neuro-muscular dissociation or selective depression of function in the bundle by pressure. Clerc[10] has suggested that in such cases all the vagal fibres are not situated in the bundle and may reach the ventricle by another route. Of recent years the experimental work of Lewis[11] has dominated all avenues of thought upon the subject of disturbances and dissociations of auricular and ventricular rhythms.

The relationship of cerebral anæmia to certain cerebral symptoms, such as epilepsy, was stressed by Russell[12] in the Goulstonian Lectures in 1909. The experimental work on this subject has been reviewed by Muskens[13] recently. He points out that, apart from intermission of the heart beat, an insufficient supply of oxygen to the brain may be a potent factor in the production of convulsions, an important point which must not be forgotten. He further shows that two factors are concerned in cases of cerebral anæmia which manifest epileptiform convulsions: (*a*) the hypoxhæmy, (*b*) the predilection all fit phenomena show for the moment of transference from the conscious to the unconscious state.

Considering next the pathological lesions found at *post mortem* examination, Sir Arthur Keith in 1909[14] showed in a review of twenty-one cases of heart block that a pathological break of continuity occurred in only eight of them. He divides the twenty-one cases into five different groups: (i) gummata of the bundle, (ii) lesions of the bundle associated with arterio-sclerosis, (iii) pathological lesions of the bundle in patients who may have had rheumatic endocarditis, (iv) conditions in which there was fatty infiltration of the junctional tissue, (v) conditions in which the junctional tissue was damaged by acute inflammation.

He also states that in some patients not manifesting any block he has found as much damage to the junctional tissue as in many in whom there were signs of complete heart block.

Numerous cases have been reported since then in which no lesion of the bundle has been demonstrated. In some of these demonstrable lesions of the coronary arteries or their ramifications have been found. The normal vascular supply to the neuro-muscular tissue has been minutely described by Gross[15] who has shown also the variations which may be found therein. Willius,[16] on the other hand, has shown the development of complete heart block following coronary thrombosis confirmed by *post mortem* examination. Gallavardin[17] has described similar cases in which Stokes-Adams syndrome was associated with complete heart block after coronary thrombosis. Robinson and Hermann[18] have shown clinical confirmation of Lewis's experiments in reporting cases showing a high rate of ventricular tachycardia following coronary occlusion. Geraudel[19] also has recently shown thrombosis of part of the coronary system in three patients with complete block in whom no lesion of the bundle could be found on serial section. Vaquez[20] has stressed the importance of the finding of Stokes-Adams syndrome and complete block in patients with no lesion of the bundle of His and expresses the opinion that variation in the vascular supply to the bundle may be the basis of many disturbances of rhythm.

In considering the differing types of auriculo-ventricular dissociation Crawford[21] suggests that from the behaviour of many types of partial block it would seem more probable that the agent, whatever it may be, is inconstant in action and with its removal the bundle may recover function perfectly.

Donzelot[22] sees an analogy to the varying block produced by faradization of the vagus in the variable dissociation produced by toxic products and variations in ischæmia of the bundle. The depression of function in the bundle produced by digitalis has recently been shown to be due to a prolongation of the refractory period of the node.[23]

The paroxysmal group is considered by Carter and Dieuaide[24] to be produced by a progressive anatomical lesion of the auriculo-ventricular bundle. They suggest "that there exists a large reserve in the conducting capacity of the auriculo-ventricular bundle which may be seriously encroached upon before conduction is measurably impaired, while some subtle local circulatory deficiency or temporary increase in vagal activity may result in the failure of the few remaining fibres to function."

It is not difficult to conceive why such patients with partial block should suffer from transient attacks of cerebral anæmia. The ventricle, changing its response to auricular impulses or initiating an idio-ventricular rhythm, is liable to cease function for a short period until the new type of rhythm is established.

The mechanism of disturbed ventricular action in complete auriculo-ventricular dissociation is, however, somewhat more difficult to explain. Herapeth[25] recently reviewed this aspect of the subject and considered the various changes in pulse rate prior to and following upon the syncopal attacks in Stokes-Adams syndrome. I have been able to collect from the literature eight cases in which electrocardiographic records were made during syncopal attacks. Cases reported by Wiltshire[26] and Strauss[27] have shown ventricular standstill with no previous change in type of ventricular complex nor in ventricular rate.

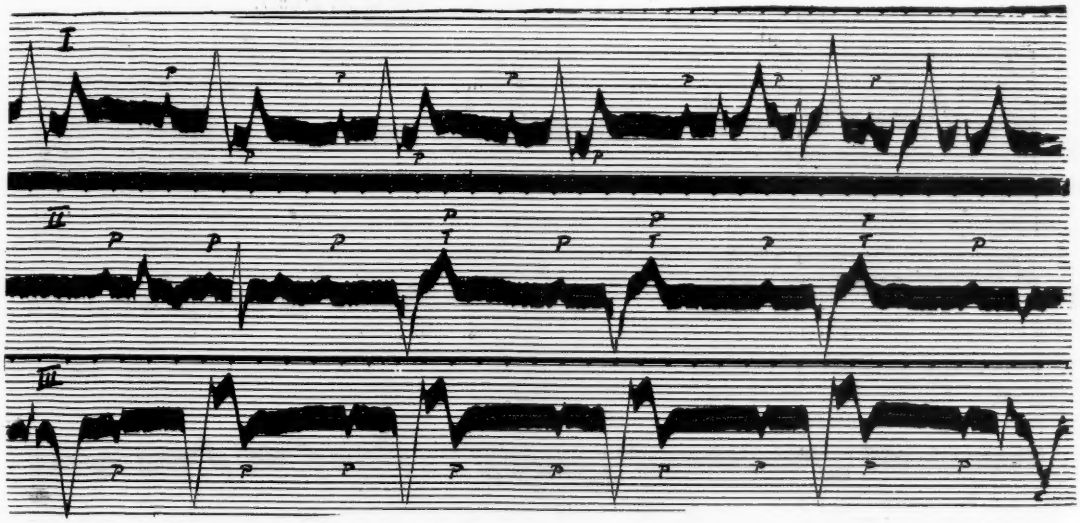

FIGURE I (Case VI), January 8, 1923.

A 2:1 type of dissociation with extra systoles occurring in Lead I. The first three ventricular complexes in Lead II are totally different each one from the other. In this and all subsequent electro-cardiograms the time marking is in one-fifth of a second.

Cases have been reported by Herapeth and by Gager and Pardee[28] in patients who have shown a quickening of ventricular rate and a probable change in the site of origin of the ventricular beat as the complexes have completely altered in type during the period of acceleration. In each instance this phase has been followed by a ventricular standstill. Following the attack in each patient there was variation in type of the ventricular complexes suggesting a conflict between the main ventricular pacemaker and an ectopic focus or foci in another part of the ventricular muscle.

Cases recorded by Allan[29] and Scott[30] have shown paroxysmal ventricular tachycardia with a degree of retrograde conduction in the bundle of His and associated syncopal attacks.

Levine and Matton[31] and Kerr and Bender[32] have recorded cases in which ventricular fibrillation occurred; both patients recovered, the former aided by intracardiac injection of adrenalin. Ventricular fibrillation is also reported by Hoesslin[33] and Gallavardin and Berard,[34] but no electrocardiographic evidence is given.

Case XIII of this series shows an apparent slowing of the ventricular rate during a syncopal attack with complete change in type of ventricular complex on recovery, but unfortunately the onset of the attack is not shown.

Although the number of cases so far reported is too small to discuss in detail, it would appear that the disturbance of ventricular action in auriculo-ventricular dissociation is by no means always of the same origin or of the same type. The occurrence and significance of varying ventricular complexes in complete block has recently been studied by Gilchrist and Cohn. They consider it is probably due to a change in leadership of the predominant pacemaker from one side of the heart to the other.

Arguing from *post mortem* evidence Geraudel[35] has propounded a theory of a separate centre for ventricular action. He considers that it is excited rhythmically by the blood flow in certain of the coronary vessels and that disease of these vessels is associated with lowering of the rate of ventricular action. This theory has been destructively criticized by Mahaim.[36]

Kaufmann and Rothberger[37] in studying certain forms of extrasystoles have described the parasystolic phenomenon. They have suggested that this is due to the interplay of two separate cardiac rhythms which arise from separate centres. They have advanced this theory to explain the coupling of beats which occurs in digitalis poisoning. No study of auriculo-ventricular dissociation based upon this conception has been published as yet.

DEFINITION.

Before considering any clinical material it is necessary to inquire further into what is implied by the term Stokes-Adams syndrome in order to establish a basis for discussion. The matter presents no little difficulty. Adams and Stokes presented certain conditions which manifested peculiar and previously undescribed features, but they neither defined a syndrome nor did they more than hazard an explanation of their facts. Moreover, it was not until fifty years later that their names were associated for the first time by Huchard.[38] He commences a fine clinical description of a group of patients thought to be suffering from bulbar arteriosclerosis in the following way:

Dans cette forme, les battements du coeur sont très lents, les pulsations radiales peuvent s'abaisser jusqu'a trente, vingt ou même cinquante par minute et s'accompagner souvent d'attaques syncopales et épileptiformes triade syndromique signalée par Adams dès 1827 ensuite par Stokes d'où le nom de maladie d'Adams ou de Stokes-Adams que je propose de lui donner.

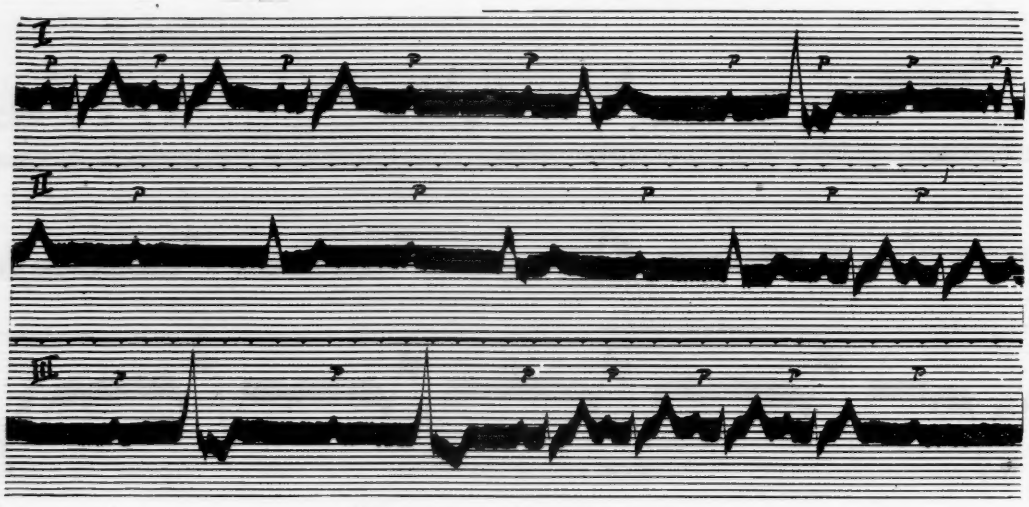

FIGURE II (Case VI), January 19, 1923.
This shows rapid alternation in degree of dissociation occurring in all three leads; a period of complete association is seen in Lead III.

Since that time there has been a tendency to apply the term to a wider group of clinical phenomena and I am unable to find any adequate and comprehensive definition. Lewis states:

> There is no agreed definition of the term, neither is the precise form of the fits described by Adams and Stokes known, however probable it be that their patients suffered from heart block;

and continues:

> there are many conditions in which a continued slow action of the pulse combines, with fainting or epileptic attacks, to form a clinical syndrome with which the names of Adams and Stokes are often associated.

Certainly in studying the literature one cannot but be struck by the diversity of conditions which are now considered to be included in the Stokes-Adams syndrome.

In 1906 Hay[39] reported a case as a form of Stokes-Adams syndrome in which storms of extra systoles produced a weakness of the pulse and convulsive seizures in a patient who had suffered from complete heart block for months. Similar conditions already referred to, described by Levine and Matton and others, would suggest that slowness of the ventricular rate is not always an essential factor in the modern conception of the syndrome.

Further the occurrence of paroxysmal attacks of tachycardia with auriculo-ventricular dissociation causing syncope in patients who at other times show a normal cardiac action, would suggest that a permanent dissociation is not essential.

Again dissociation of any degree would not seem necessary. I shall describe a case in which syncopal attacks have occurred in a patient with sino-auricular block with a slowing of the entire heart and occasional syncopal seizures. Tumours involving the vagus nerve as reported by Boot,[40] Cassidy,[41] Sicard[42] and others have also been described as producing the syndrome.

There would appear to be one factor which is common to all types and that is such a disturbance of cerebral circulation as to produce syncope or convulsive seizures. This failure which is invariably manifested with suddenness, may arise from a variety of causes either in the heart or remote therefrom, but all resulting in such a serious disturbance of ventricular output as to prevent the maintenance of an adequate blood supply to the brain. Although the occurrence of symptoms of cerebral anæmia associated with a persistent slow pulse will accurately cover the majority of cases, I feel that the syndrome should be defined so as to embrace a wider conception of the underlying principles and to include all the varieties which have been considered.

I would therefore submit as a tentative definition: The Stokes-Adams syndrome comprises a sudden failure in the maintenance of an adequate cerebral circulation of such degree as to produce syncopal apoplectiform or epileptiform seizures, such failure arising as a direct consequence of a

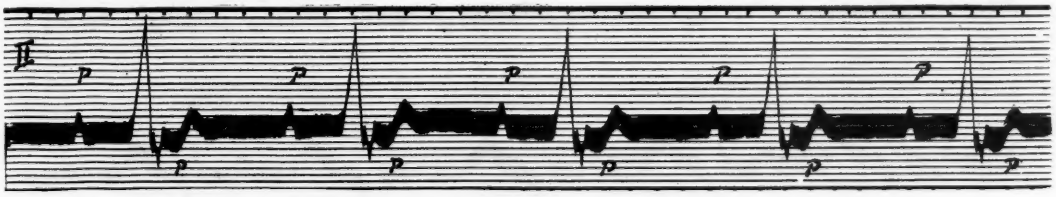

FIGURE III (Case VI), January 20, 1923.
Showing a 2:1 auriculo-ventricular dissociation.

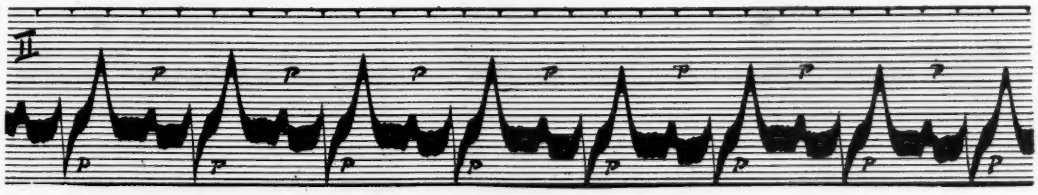

FIGURE IV (Case VI), March 5, 1923.
Showing a tachycardia with 2:1 auriculo-ventricular dissociation.

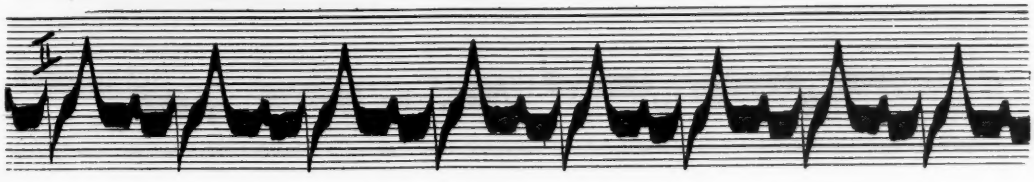

FIGURE V (Case VI), May 23, 1923.
Showing a further phase of a similar type of tachycardia.

lessened cardiac output from a grave disturbance of ventricular action.

CLINICAL CONSIDERATIONS.

In an attempt to ascertain the relationship which exists between Stokes-Adams syndrome and auriculo-ventricular dissociation, I have collected from the records of Dr. Strickland Goodall twenty-seven cases of heart block. In every one of these the existence of dissociation has been repeatedly confirmed by electrocardiograms.

Thirteen of the twenty-seven patients gave evidence of the Stokes-Adams syndrome.

Two further cases in which in my opinion the syndrome was due to another cause, will be described later.

Of the twenty-nine patients eleven are known to have died, six cannot be traced, while twelve have been observed by me.

Notes of the clinical conditions are reproduced at the end of this article.

I propose to consider first the twenty-seven cases in which auriculo-ventricular dissociation was manifested. Certain factors common to the group as a whole will be dealt with and points of interest in individual cases will be discussed.

Before commencing any discussion on dissociation, it will be convenient to divide it into two types. The first is variable or intermittent dissociation. This group comprises all those cases in which from time to time there has been a difference or variation in the degree of dissociation. All the patients whose condition is placed under this heading in my series have at one time or another shown a phase of complete dissociation of auricles and ventricles.

The second group is constant or continuous dissociation. This group comprises those cases in which no variation in degree of dissociation has occurred over the period of observation; with one exception (a constant 2:1 block) complete dissociation of auricles and ventricles has been present in every case in my series.

It is important to establish and appreciate the distinction between the terms variable and constant which apply to dissociation considered over a period of time, and the terms partial and complete which apply to any single consideration of dissociation.

Auriculo-ventricular dissociation is a rare manifestation of cardiac disease. As a rough estimate of its frequency, these twenty-seven patients are collected from a series of approximately nine

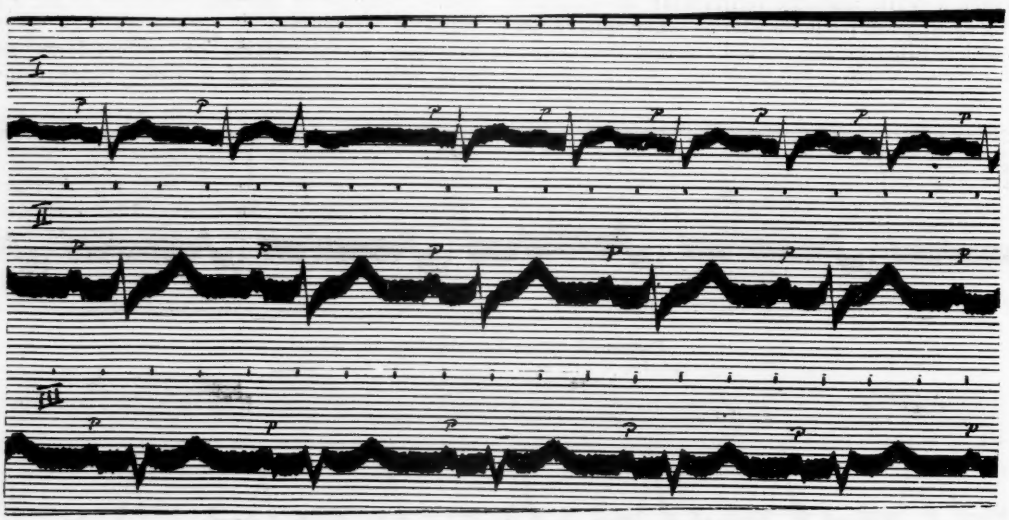

FIGURE VI (Case VI), September 3, 1923.
Showing a period of normal associated rhythm. These plates are taken over a period when this patient was having repeated syncopal attacks, due no doubt to disturbance of ventricular rhythm and affection of the ventricular myocardium by what has been aptly described as a "shifting myocarditis." With antisyphilitic treatment an associated rhythm continued for over four years. The condition then relapsed.

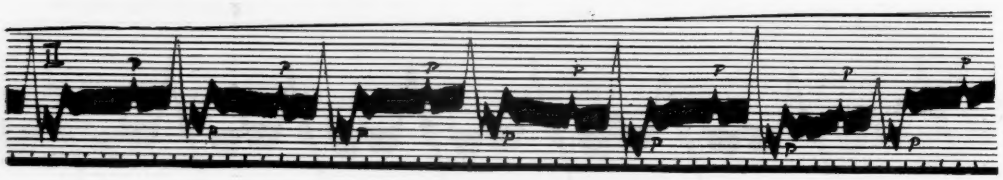

FIGURE VII (Case VI), March 23, 1928.
Showing a phase of 2:1 dissociation since the relapse.

thousand patients, all of whom attended hospital under the care of Dr. Strickland Goodall between 1918 and 1928.

Ætiological Factors.

I propose now to consider the ætiological factors of the group.

Sex.

Twenty patients were males; seven patients were females.

Age.

The youngest patient was twenty-two years when first observed. The oldest patient was seventy years when first observed. The age incidence is shown in Table I.

Familial Factors.

No case of congenital heart block is included, but one patient (No. X) had a son who suffered from complete heart block. Dr. Strickland Goodall has examined three generations from this family and has found complete heart block in grandmother, mother and son.

Causation.

In seeking for causative factors one cannot dogmaticaly attribute dissociation to this or that associated condition. Nevertheless, it has been possible to subdivide the cases into three main groups: syphilitic, toxic, degenerative.

The relationship between these groups and the several factors is shown in Table I.

Of the four syphilitic patients three had acquired syphilis and their average age was forty-three years. One patient had congenital syphilis and was twenty-two years of age. Three patients manifested the Stokes-Adams syndrome.

Of the five patients with toxic conditions the average age was thirty-five years. Four of these patients had suffered from severe acute influenza not more than six months prior to the development of heart block. One patient from the nature of onset and course apparently suffered from an acute infection of unknown ætiology. Three of this group manifested the Stokes-Adams syndrome.

TABLE I.

Twenty-seven Cases of Auriculo-ventricular Dissociation.

Age.	Sex		Causative Factors.			Type of Dissociation.		Stokes-Adams Syndrome
	Male.	Female.	Syphilis.	Toxic.	Degenerative.	Variable.	Constant.	
21–30	1	3	1	2	1	1	3	2
31–40	4	1	1	2	2	1	4	2
41–50	4	1	1	1	3	2	3	4
51–60	6	1	1	—	6	2	5	3
61–70	5	1	—	—	6	2	4	2
	20	7	4	5	18	8	19	13

Of the eighteen patients with degenerative conditions the average age was fifty-four years, but I have divided this group into two.

Of the six patients under fifty years of age, four had suffered from a severe infection many years previously and had associated endocardial damage (two rheumatic fever, one scarlet fever and one influenza). In two no cause was apparent.

There were twelve patients over fifty years of age and in all there was some definite clinical evidence of myocardial degeneration, endocardial disease or arterio-sclerosis. Seven of this group of eighteen manifested the Stokes-Adams syndrome.

The relationship between these groups and type of dissociation and age is shown in Table II.

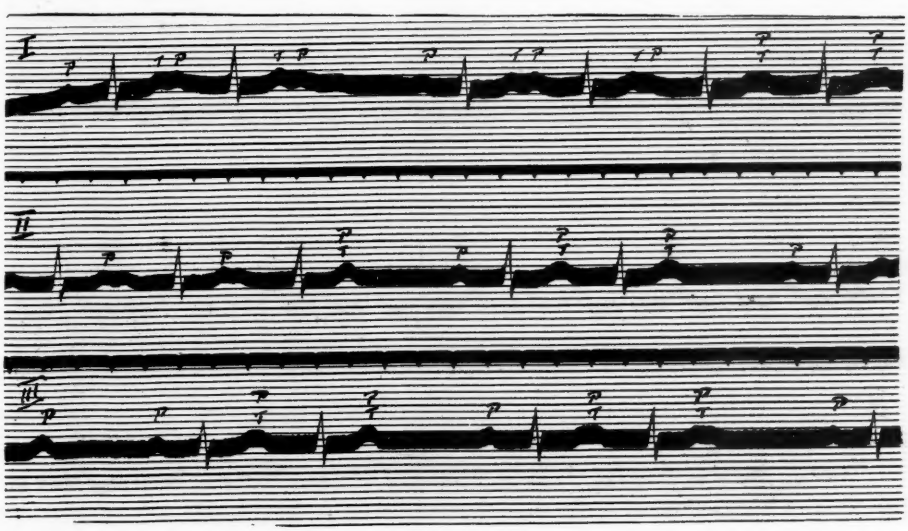

FIGURE VIII (Case XXII), March 22, 1923.
Showing complete auriculo-ventricular dissociation occurring shortly after severe influenza.

TABLE II.

Thirteen Cases of Auriculo-ventricular Dissociation showing Stokes-Adams Syndrome

Age.	Male.	Female.	Syphilis.	Toxic.	Degenerative.	Variable.	Constant.
21–30	1	1	1	1	—	1	1
31–40	1	1	1	1	—	1	1
41–50	3	1	1	1	2	3	1
51–60	3	—	—	—	3	1	2
61–70	1	1	—	—	2	—	2
	9	4	3	3	7	6	7

Relationship of the Type of Block to Incidence of the Stokes-Adams Syndrome.

Of eight patients with variable auriculo-ventricular dissociation, six manifested the Stokes-Adams syndrome. Of these six persons, the average age was forty-one years. The condition in three was toxic, in one was syphilitic and in two was degenerative.

Of nineteen patients with constant auriculo-ventricular dissociation, seven manifested the Stokes-Adams syndrome. Of these seven the average age was fifty-eight years. The condition in two was syphilitic and in five was degenerative.

Although the number is too small from which to draw any definite conclusions, it would seem that Stokes-Adams syndrome is very frequent in variable dissociation, while only one-third of the patients with constant dissociation manifests it.

I shall refer to the age difference between the two groups in a later section.

Symptomatology.

I have investigated the symptomatology of the group with a view to ascertaining whether, apart from syncopal attacks, any other difference exists between those patients with Stokes-Adams syndrome and those without. I have been unable to distinguish between the two types.

The results are given in Table III.

TABLE III.

Symptom.	Fourteen Patients without Stokes-Adams Syndrome.	Thirteen Patients with Stokes-Adams Syndrome.	Total.
Dyspnœa	13	11	24
Vertigo	10	8	18
Pain	9	6	15
Palpitation	6	3	9
Exhaustion	2	2	4

Clinical Condition of Cardio-Vascular System.

The average pulse rate for the whole series, based upon the radial pulse at first examination, was 38. At some time or other every patient in the series has shown some variation of rate. In the series of constant and complete block the highest recorded pulse rate was in the patient Case XIII, whose ventricular rate reached 62 per minute while completely dissociated from the auricular rate. The lowest recorded rate was eight per minute in the patient Case III, while in a fit.

In Table IV is shown the average blood pressure, systolic and diastolic, in sixteen patients.

TABLE IV.

Average Pressure.	Eight Patients without Stokes-Adams Syndrome.	Eight Patients with Stokes-Adams Syndrome.	Total.
	Millimetres.	Millimetres.	Millimetres.
Systolic	160	162	161
Diastolic	73	78	75
Pulse	87	84	86

Palpable evidence of arterio-sclerotic changes in peripheral vessels was found in sixteen patients.

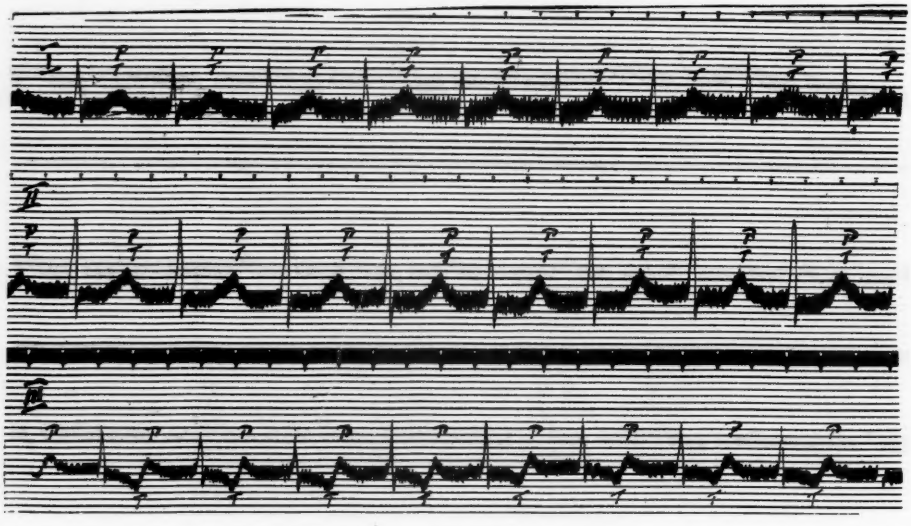

FIGURE IX (Case XXII), May 24, 1923.
Showing a tachycardia with superimposed *T* and *P* waves, but no dissociation.

TABLE V.

State of Arteries.	Fourteen Patients without Stokes-Adams Syndrome.	Thirteen Patients with Stokes-Adams Syndrome.	Total.
Arterio-sclerotic change ..	9	7	16
No arterio-sclerotic change	5	6	11

The condition of the heart in the twenty-seven patients is set out in Tables VI and VII.

TABLE VI.

Clinical Enlargement.	Fourteen Patients without Stokes-Adams Syndrome.	Thirteen Patients with Stokes-Adams Syndrome.	Total.
To left	8	7	15
To right and left	4	2	6
No enlargement	2	4	6

TABLE VII.

Endocardial Disease.	Fourteen Patients without Stokes-Adams Syndrome.	Thirteen Patients with Stokes-Adams Syndrome.	Total.
Aortic incompetence and mitral incompetence ..	3	4	7
Mitral stenosis and mitral incompetence	1	—	1
Auricular fibrillation ..	2	—	2
Mitral incompetence ..	6	7	13
No apparent disease ..	2	2	4

Thus no clinical distinction can be drawn from the symptomatology or the examination of the cardio-vascular system between patients suffering from auriculo-ventricular dissociation who manifest Stokes-Adams syndrome and those who do not, apart from variations in degree of block which may be apparent on different occasions.

The high pulse pressure in these patients and the frequency of mitral regurgitation are points of clinical interest.

The Nervous Symptoms in Stokes-Adams Syndrome.

Among the thirteen patients under consideration ten had no gross abnormality of the nervous system, one had bilateral deafness and other stigmata of congenital syphilis, one had signs of an old cerebral thrombosis, in one a cerebral thrombosis developed while he was attending hospital. No patient presented any family history of epilepsy.

It is difficult to state with any degree of exactness the type of attack in most of these patients. A fine distinction exists between the vertigo complained of by patients with the various types of cardiac lesions and the earliest signs of cerebral anæmia encountered in persons with heart block. At the risk of excluding a certain number I have included in the category of Stokes-Adams syndrome only those in whom there has been a loss of consciousness for a period of some seconds or longer or those in whom definite convulsive seizures have occurred. To the first type the terms syncopal or apoplectiform have been applied and to the second type the term epileptiform has been given.

Owing to the difficulty of judging the type of attack from descriptions by patients' friends in some instances, the following figures can be regarded only as a very rough approximation. Syncopal or apoplectiform attacks occurred in nine patients. Epileptiform attacks occurred in four patients.

In two patients, both of whom suffered from epileptiform attacks, an aura preceded some of the attacks; in one it was visual in type, in the other epigastric. In several others a vague consciousness of disturbed cardiac action preceded an attack. In most, however, no warning occurred. In a few the precipitation of an attack followed undue exertion, but in others no causative agent was

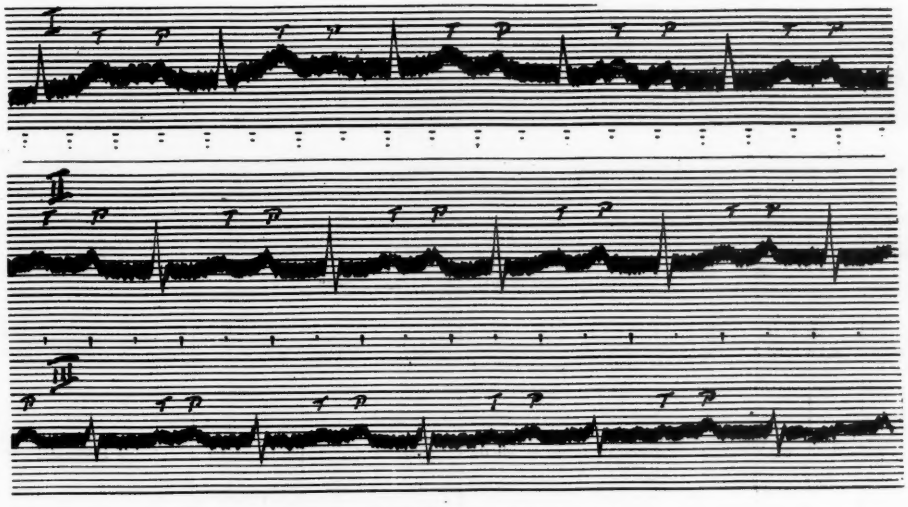

FIGURE X (Case XXII), October 12, 1928.
Showing a normal associated rhythm with a lengthened *P-R* interval and general flattening of the curve.

TABLE IX.

No.	Initials.	Sex	Age	Response to Wassermann Test.	Past History	First Observed.	Symptoms.						Duration of Stokes-Adams Syndrome.	Pulse.	Blood Pressure.	
							Fit.	Dyspnœa.	Vertigo.	Pain.	Palpitation.	Exhaustion.			Systolic.	Diastolic.
I	F.L.	F.	47	0	Severe influenza at age of 19; heart disease for 18 years.	1918	+	+	+				18 years	32	168	
II	L.W.	F.	37	+	Enteric fever as child; slow pulse for 10 years.	1918	+	+			+	+	10 years	34		
III	C.H.	M.	27	0	Severe influenza 6 months previously.	1920	+	+	+				6 months	48	132	
IV	A.W.	M.	52	0	Nothing relevant	1920	+	+	+			+	8 years	32	170	75
V	J.N.	M.	31	0	Demobolized A1; severe influenza 1919 and in 1921	1922	+	+	+				1 year	32	130	80
VI	A.G.	M.	42	+	Nothing relevant.	1923	+		+				1 year	40	130	50
VII	E.M.	F.	22	+	Scarlet fever as a child; stigmata of congenital syphilis.	1923	+	+	+				7 years	36		
VIII	G.J.	M.	42	0	Nothing relevant.	1924	+						Few days	40	140	75
IX	J.M.	M.	43	0	Measles as a child.	1924	+	+					1 year	34	130	50
X	F.P.	F.	61	0	Nothing relevant.	1924	+	+				+	2 years	28	228	85
XI	S.H.	M.	70	0	Nothing relevant	1925	+	+	+			+	1 year	35	170	90
XII	F.McG.	F.	51	0	Rheumatic fever at 15 years and twice later; severe pyorrhœa.	1925	+	+		+	+	+	2 years	70	180	110
XIII	H.O'C.	M.	56	0	Growing pains in childhood; A1 in 1915 severe influenza in 1918	1926	+	+	+				2 years	32	135	90
XIV	H.B.	M.	59	0	Influenza 15 years before, malaria, dysentery at war	1927	+	+	+	+		+	5 months	48	220	110
XV	J.D.	M.	61	0	Scarlet fever as child; influenza years before.	1927	+	+	+	+	+		1 year	44	160	100

TABLE IX (*Continued*).

Arteries.	Heart.	Attacks.	Electrocardiogram.	Type.	Progress.	Probable Ætiology.
Not thickened.	Enlarged to left and right, mitral regurgitant murmur.	Apoplectiform, falls suddenly, consciousness lost for some seconds.	Complete *a-v* dissociation: ventricular complexes show left preponderance.	Constant complete block.	Death sudden in fit 1919.	Degenerative, post-influenzal.
Not thickened.	Not enlarged, no adventitiæ.	Apoplectiform, severe attacks for years; 6 months before death developed cerebral thrombosis.	Complete *a-v* dissociation, left ventricular extrasystoles; no gross change in curve in 10 years.	Constant complete block.	Death sudden in fit 1926.	Syphilitic.
Thickened	Enlarged to left, aortic regurgitation, mitral regurgitation.	Apoplectiform; duration variable up to ½ hour, but no convulsions; attacks vary in severity.	Variable dissociation 2 : 1 and complete; gradual development of right branch block.	Variable block.	Death 1924.	Influenza, toxic.
Thickened.	Enlarged to left, mitral regurgitation.	Apoplectiform, lasting a few minutes; has had attacks for 8 years; now very frequent.	Complete *a-v* dissociation; curve becoming isoelectric.	Constant complete block.	Observed; now feeble and unable to get about.	Degenerative.
Not thickened.	Enlarged to left, mitral regurgitation.	Apoplectiform; short attacks of loss of consciousness, lasting few seconds.	Variable dissociation 3 : 1, 2 : 1 complete.	Variable block.	Sudden death 1922.	Influenza, toxic.
	Enlarged to left, aortic regurgitation, mitral regurgitation.	Epileptiform; variable type suggests *petit mal* and major epilepsy; after treatment was free from attacks for 5 years; now has occasional syncopal attacks.	Extremely variable dissociation, then associated rhythm for 5 years, now variable 2 : 1 block.	Variable block.	Observed; cured by anti-syphilitic treatment, relapse after 5 years, now improving.	Syphilitic.
Not thickened.	Not enlarged, mitral regurgitation.	Epileptiform and momentary syncopal attacks.	Complete *a-v* dissociation, at times coupling and nodal extrasystoles.	Constant complete block.	Sudden death 1925.	Syphilic (congenital).
Not thickened.	Not enlarged, no adventitiæ.	Epileptiform and apoplectiform; visual aura before some attacks; had repeated attacks for some weeks.	Variable *a-v* dissociation, complete reverting to variable types of association and later normal beats.	Variable block.	Observed; no attacks over period of 3 years; associated cardiac action.	Acute infective, toxic.
Thick, tortuous.	Enlarged to left, aortic regurgitation, mitral regurgitation.	Syncopal attacks without apparent cause, later apoplectiform seizures.	Normal curve, later showing variable dissociation and now for 2 years constant 2 : 1 and right branch block.	Variable block.	Observed; fits corresponding to period of variable block; free from same for 2 years.	Degenerative.
Thick.	Enlarged to left, aortic regurgitation, mitral regurgitation.	Apoplectiform attacks, unconscious for seconds; son has similar attacks.	Complete *a-v* dissociation, runs of left ventricular extrasystoles.	Constant complete block.	Untraced.	Degenerative.
Thick.	Enlarged to left, mitral regurgitation.	Apoplectiform; short duration.	Complete *a-v* dissociation and left branch block.	Constant complete block.	Observed; well and free from fits for 12 months.	Degenerative.
Not thick.	Enlarged to left, no adventitiæ.	Apoplectiform; duration ½ hour during attack of tachycardia.	Normal associated rhythm and attacks of paroxysmal tachycardia; ventricle rate = 240 per minute.	Paroxysmal tachycardia.	Untraced since 1926.	Degenerative.
Thick.	Not enlarged.	Epileptiform; usually epigastric aura before convulsion; attacks varied in type and severity.	Variable dissociation from complete to associated rhythm, left ventricular preponderance.	Variable block.	Death from cholecystitis and peritonitis; *post mortem* confirmed cardiosclerosis and coronary disease.	Degenerative.
Thick.	Much enlarged to left, mitral regurgitation.	Apoplectiform, duration few seconds; attacks becoming less frequent of late; exertion precipitates attack.	Complete *a-v* dissociation, with early left bundle block; now shows no dissociation, but right bundle block developed.	Complete constant block, reverting to associated rhythm.	Observed; is well and able to get about in comfort.	Degenerative and possibly a toxic factor.
Not thickened	Not enlarged, no adventitiæ.	Often vertiginous, but has had several syncopal attacks in which he has lost consciousness for some seconds.	No *a-v* dissociation, cardiac complexes, physiological sino-auricular block.	Sino-auricular block.	Observed; *in statu quo*; is able to get about, but not to work.	Degenerative.

TABLE X.

No.	Initial.	Sex.	Age.	Response to Wassermann Test.	Past History.	First Observed.	Symptoms.					Duration of Symptoms.	Pulse.	Blood Pressure.	
							Dyspnœa.	Vertigo.	Palpitation.	Exhaustion.	Pain.			Systolic.	Diastolic.
XVI	F.McG.	F.	27	0	Severe scarlet fever at 4 years.	1918	+		+			8 years	36		
XVII	H.G.	M.	40	0	Enteric fever and dysentery at war; influenza 1918.	1918	+	+	+			2 years	20	130	60
XVIII	T.C.	M.	64	0	Nothing relevant	1920	+	+			+	8 months	40		
XIX	T.P.	M.	33	0	Nothing relevant, except rheumatic fever as a child.	1920	+	+			+	4 months	40	160	90
XX	J.S.	M	63	0	Severe influenza three times years before.	1921	+	+	+		+	2 years	34	200	90
XXI	F.D.	M.	65	0	Enteric fever 30 years before.	1923	+	+				2 years	50	135	65
XXII	F.E.	F.	30	0	Severe influenza 3 months and 2 years previously.	1923		+	+	+		9 months	50	125	60
XXIII	C.B.	M.	33	0	Rheumatic fever as child.	1924	+	+			+	2 years	32		
XXIV	W.D.	M.	69	0	Severe influenza in 1918.	1924	+				+	1 year	38	170	50
XXV	F.H.	M.	60	0	Rheumatic fever years before.	1924	+	+		+	+	1 year	40		
XXVI	A.J.	M.	59	0	Rheumatic fever at 25 years and at 42 years.	1925	+	+		+	+	2 months	40		
XXVII	C.B.	F.	58	0	Nothing relevant.	1926	+	+	+			1 year	24	195	70
XXVIII	H.B.	M.	48	0	Nothing relevant.	1927	+		+		+	2 years	46	140	90
XXIX	W.H.	M.	52	+	Malaria 1.14.	1927	+	+			+	2 years	32	144	60

TABLE X (*Continued*).

Arteries.	Heart.	Electrocardiogram	Type.	Progress.	Probable Ætiology.
Thick.	Mitral stenosis and regurgitation, enlarged to right and left.	Complete *a-v* dissociation.	Constant complete block.	Sudden death 1922.	Degenerative, post scarlet fever.
Thick, tortuous.	Enlarged to left, mitral regurgitation.	Complete *a-v* dissociation.	Constant complete block.	Untraced after 1921.	Influenza, toxic.
Thick.	Enlarged to left, no adventitiæ.	Very variable *a-v* dissociation, with early and developed right branch block; occasional left ventricular tachycardia.	Variable block.	No Stokes-Adams syndrome over 6 years; untraced after 1927.	Degenerative.
Not thick.	Enlarged to left and right, auricular fibrillation.	Complete *a-v* dissociation, with isoelectric fibrillation; idioventricular rhythm and left ventricular extrasystoles.	Constant complete block.	Death 1922.	Degenerative, post rheumatic.
Thick.	Enlarged to left, mitral regurgitation.	Complete *a-v* dissociation.	Constant complete block.	Untraced from 1924.	Degenerative.
Thick.	Enlarged to left, auricular fibrillation.	Complete *a-v* dissociation; auricular fibrillation developed while under observation.	Constant complete block.	Death 1924.	Degenerative.
Not thickened.	Enlarged to left, no adventitiæ.	Complete *a-v* dissociation; later reversion to associated rhythm and tachycardia; now shows associated rhythm PR = 0·35".	Constant complete block, reversion to associated rhythm.	Observed; well and free from symptoms for 3 years.	Influenza, toxic.
Not thickened.	Enlarged to left, mitral regurgitation.	Complete *a-v* dissociation; no change over period of 4 years.	Constant complete block.	Observed; severe vertigo recently, but no Stokes-Adams.	Degenerative.
Thick.	Enlarged to left, no adventitiæ.	Complete *a-v* dissociation.	Constant complete block.	Observed; well, no increase of symptoms.	Degenerative.
Thick, tortuous.	Enlarged to right and left, aortic regurgitation, mitral regurgitation.	Complete *a-v* dissociation with gradually decreasing voltage during observation.	Constant complete block.	Death 1925; carcinoma of œsophagus.	Degenerative.
Thick.	Enlarged to right and left, aortic regurgitation, mitral regurgitation.	Variable dissociation 3 : 2, 2 : 1, complete, settling at 2 : 1 rhythm.	Variable block.	No Stokes-Adams syndrome; some improvement under treatment; death 1926.	Degenerative.
Not thickened.	Enlarged to right and left, aortic regurgitation, mitral regurgitation.	Complete *a-v* dissociation; no change in type of curve under observation.	Constant complete block.	Observed; has attacks of vertigo; no Stokes-Adams syndrome; otherwise well.	Degenerative.
Not thickened.	Not enlarged, no adventitiæ.	Constant 2 : 1 *a-v* dissociation; no change under observation in 18 months.	Constant 2 : 1 block.	Untraced for past 10 months.	? Degenerative.
Thick.	Enlarged to left, mitral regurgitation.	Complete *a-v* dissociation.	Constant complete block.	Observed; well, no change in symptoms.	Syphilitic.

apparent. The longest attack authentically observed occurred in the patient Case XIII, when the patient remained unconscious for over half an hour, but no convulsions occurred. In others repeated short attacks, epileptiform in character, occurred at intervals with but transient loss of consciousness. The frequency of the attacks varied from repeated attacks in one day, as in Case VI just mentioned, to isolated single syncopal attacks at intervals of weeks, months or even years, for example Case I. I have not sufficient evidence to make any statement with regard to the relationship, if any, between the pulse rate immediately before an attack and the duration thereof.

Clinical Course.

The progress of all the cases of auriculo-ventricular dissociation will first be considered. Of the twenty-seven patients whose condition is described, eleven are known to have died, two are known to have recovered, three cannot be traced and eleven have been personally observed.

The relationship of a group of the eleven who died, to the various ætiological factors already mentioned, can best be shown by means of a table (see Table VIII).

It would thus appear that the outlook for patients who manifest the Stokes-Adams syndrome, is worse than for those who do not.

Further, those in whom there is a variable form of dissociation, are associated with a higher mortality than those with a constant form, while the gravest prognosis is attached to those with the Stokes-Adams syndrome associated with variable heart block.

The mode of death in five of the six patients who died with Stokes-Adams syndrome was sudden and in each death occurred in a syncopal attack. The sixth patient died from general peritonitis and acute cholecystitis. In one of the five who did not have Stokes-Adams syndrome, death was sudden. I have been unable to ascertain the mode of death of the other four patients.

TABLE VIII.

Total Number of Cases.	Type of Dissociation.	With Stokes-Adams Syndrome.	Without Stokes-Adams Syndrome.	Number Dead.	Ætiology.			Average Age of Death.	Average Duration After Onset of Symptoms.
					Syphilis.	Toxic.	Degenerative.		
								Years.	Years.
8	Variable	6	—	3	—	2	1	38	1½
		—	2	1	—	—	1	59	2
19	Constant	7	—	3	2	—	1	36	10
		—	12	4	—	—	4	50	6
27		13	14	11	2	2	7		

It is a significant fact that no patient is known to have died of progressive myocardial failure and only one yielded clear evidence of congestive failure at any time.

Of the eleven patients observed the condition has apparently cleared up in two; in one it cleared up for five years and then relapsed to variable block; two are seriously incapacitated, while six are comparatively well.

To summarize each case, the following are the records of the three patients who recovered.

CASE VIII.—The patient had a variable block with Stokes-Adams syndrome, infective in type, has shown no dissociation for three years and is now well.

CASE XXII.—The patient had a condition considered as a constant block occurring after influenza. He has been well and free from symptoms for three years and has a normal association, except for a prolonged *P-R* interval in electrocardiogram.

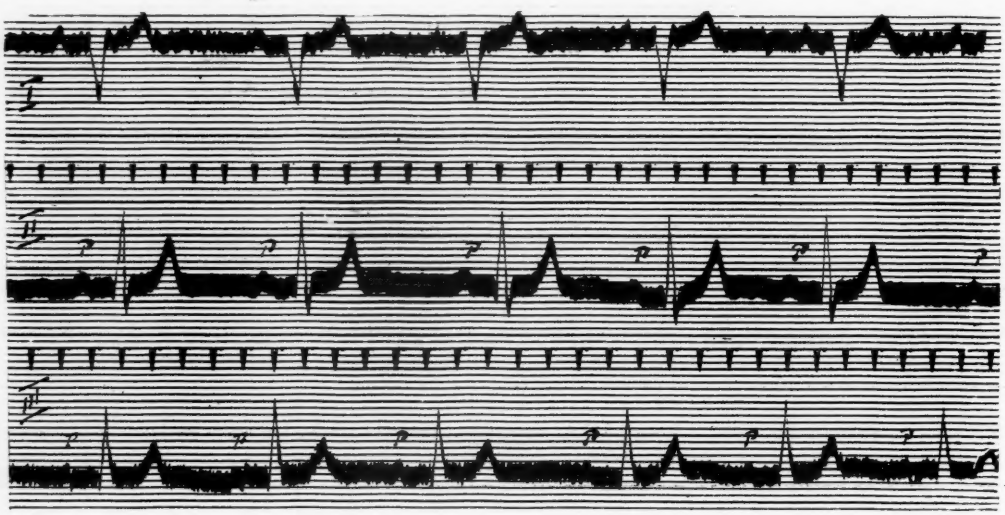

FIGURE XI (Case IX), May 9, 1925.
Showing a normal type of rhythm.

CASE VI.—The patient who suffered a relapse, had a very variable block with Stokes-Adams syndrome which responded to antisyphilitic measures, resulting in an associated rhythm. Five years later a return of symptoms and a partial heart block developed. He is now improving under further treatment.

The next two are incapacitated.

CASE IV.—This patient had a constant complete block and has been attending hospital for the past eight years; he has manifested the Stokes-Adams syndrome for that length of time. He is now feeble and has repeated syncopes and consequently is unable to get about.

CASE XXVII.—This patient also had a constant complete block and has been under observation for two years. Although no syncopal attacks have occurred within the past few months, severe vertigo has occurred at intervals.

The six patients whose condition has remained *in statu quo*, are dealt with in the next group.

CASE IX.—This patient at first had paroxysmal dissociation. Syncopal attacks occurred at intervals and normal rhythm was usually present. Later a variable dissociation occurred with further syncopes. For the past two years there has been an apparent 2 : 1 dissociation with freedom from attacks. The patient is able to do light work.

CASE XI.—This patient had a complete constant block associated with the Stokes-Adams syndrome, but following the administration of barium chloride there have been no attacks for twelve months. The patient is able to get about quite well.

CASE XIV.—This patient was thought to be suffering from a complete dissociation when first seen a year ago. At that time syncopal attacks occurred fairly often. Of late there has been freedom from them and electrocardiographic evidence shows a curious change has occurred, to be discussed later.

CASE XXIV.—This patient had a constant complete block and was under observation for four years. He did not have any syncopal attacks. At the age of seventy-three he is now able to get about without distress.

CASE XXIX.—This patient who had a constant complete block, was observed for twelve months. He has had no syncopal attacks and is able to do light work.

CASE XXVII.—This patient who had a constant complete block, has been under observation for the past two years. No syncopal attacks have occurred, but the patient becomes vertiginous on any undue exertion.

No member of this group of six can be said to be capable of leading an active, strenuous life, but realizing the necessity of a reduction in their activities, they all lead a comparatively comfortable, but somewhat sheltered existence.

Electrocardiographic Evidence.

The ultimate diagnosis in every case of the nature and degree of auriculo-ventricular dissociation present was made by electrocardiogram. In view of the controversial opinions with regard to interpretation of certain findings held by various writers on the subject,[43] I have confined my study to those features of the curve of which the significance is more or less certain.

Fourteen of the twenty-seven patients had complete auriculo-ventricular dissociation. In twelve this was constant at all examinations, complete in eleven and 2 : 1 in one. In two it was followed by a reversion to associated rhythm without variability occurring as far as is known. Thirteen had variable auriculo-ventricular dissociation; with no exception all at one time or another had a complete dissociation.

Of associated conditions in curves of the twenty-seven patients extra systoles arising from ectopic foci were shown to occur in fourteen, left-sided preponderance occurred in eight, right branch block was found in three and left branch block was found in two.

Variations in the type of ventricular complex occurring in the same patient were found six times. The significance of this to me is not certain, but it would suggest the origin of the ventricular beats from separate parts of the ventricular conducting system or ventricular muscle, a possible manifestation of hyperirritability or exhaustion of the ventricular myocardium.

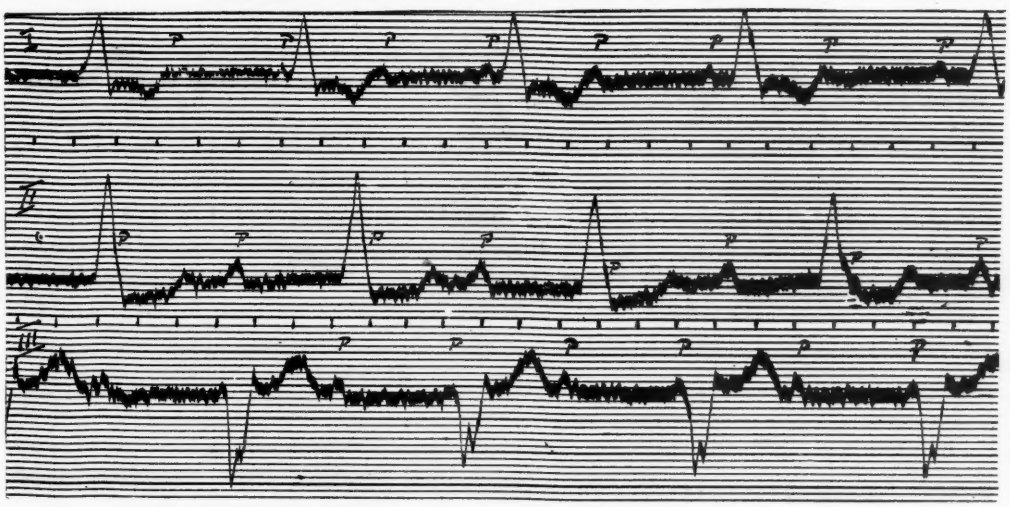

FIGURE XII (Case IX), November 5, 1928.

Showing a complete auriculo-ventricular dissociation and a right branch block. Between these two phases a paroxysmal dissociation occurred with variation in degree of the dissociation. Syncopal seizures occurred during this period. Later a complete dissociation developed with right branch block. This condition has apparently remained unchanged for the past two years. No distinct alteration has occurred in the electro-cardiogram over this period and there have been no further syncopal attacks.

It is impossible to include tracings from all the cases considered, but I have included typical electrocardiograms of certain conditions with brief notes.

I have been unable to determine any electrocardiographic feature apart from that shown by variable, as opposed to constant, dissociation which would even suggest a distinction between those patients in whom Stokes-Adams syndrome developed and those in whom it did not.

Post Mortem Evidence.

In two instances only has a *post mortem* examination been performed upon patients in this series. In each definite degenerative changes in the heart of somewhat different type were found.

CASE XIII.—Death occurred from general peritonitis and cholecystitis. Definite sclerotic changes were found diffusely scattered through the heart, but nowhere involving the bundle of His to any great extent. There was found a distinct atheroma of the aorta and particularly of the anterior descending branch of the left coronary artery, the lumen of which was almost obliterated.

CASE XXV.—Death was due to carcinoma of the œsophagus. There was a general enlargement of all chambers of the heart. The muscle was tough, the aorta was dilated and there was considerable calcification of the aortic valves and sclerosis of the surrounding parts which had involved the main bundle of His in much degenerative fibrosis.

The patient in Case XIII during life had had a variable type of dissociation, while in the patient in Case XXV the dissociation was of a constant and complete form.

Diagnosis.

In a typical case of auriculo-ventricular dissociation there will usually be little difficulty in the establishment of the diagnosis of the Stokes-Adams syndrome. The occurrence of syncopal or convulsive attacks in a patient showing severe bradycardia will be diagnostic.

The clinical picture in a well defined case will be characterized by a slow pulse with a correspondingly slow ventricular rate. The rhythm, usually regular, may at times be disturbed by extra systoles. It may be possible on auscultation during the long pauses between ventricular beats to hear faint auricular beats and I have observed this twice in my series. More frequently distinct pulsation can be observed in the veins of the neck with extra large pulsations at intervals when auricular and ventricular systole occurs simultaneously.

The failure to affect the ventricular rate in these patients by exercise is of importance and is in contrast to the sudden doubling in rate which occurs in certain persons with athlete's heart and to the rising response of the normal heart. Vagal compression, ocular pressure and the exhibition of atropine all fail to influence the ventricular rate in complete dissociation.

Difficulty may arise, however, in cases where the symptoms are less well defined. The establishment of dissociation may not be easy, apart from instrumental means. I have recently observed such a case (not included in this series) in which the ventricular rate was 68 per minute and in which the presence of complete heart block was only discovered by an electrocardiogram.

Further difficulty may arise in some cases of variable block in which one may obtain a suggestive history, but on examination a normal rhythm is found. Such cases may be diagnosed as epilepsy and their true nature may be apparent only after repeated observation. I would stress this point and quote as an example Case VII. The patient had several syncopal attacks and was then observed in hospital for some weeks, but at no time had anything but a normal rhythm. Later he developed a variable type of dissociation and had further syncopal attacks during that time and so the diagnosis was established.

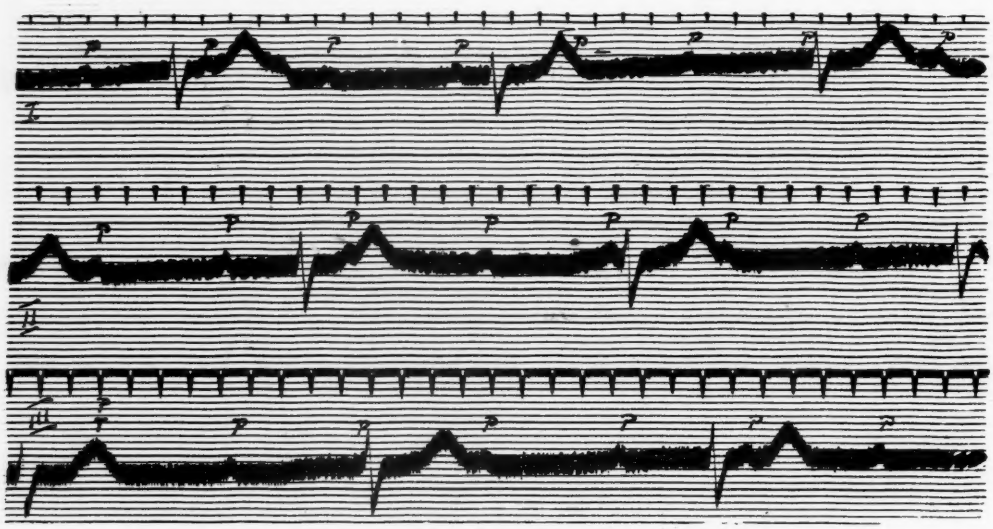

FIGURE XIII (Case X), April 29, 1924.
Showing a typical uncomplicated complete dissociation.

I would emphasize the necessity of repeated clinical and electrocardiographic examination of every patient suspected of dissociation, as it affords the only definite means of elucidating obscure forms of cardiac rhythm.

The diagnosis of Stokes-Adams syndrome arising as a result of some extracardiac cause is a controversial matter. Such conditions as tumours involving the vagus nerves are of sufficient rarity to be regarded as pathological and cardiological curiosities and will not be further considered.

Prognosis.

It is unwise to dogmatize upon such a small series as is presented here, but certain generalizations may be made. The condition of auriculo-ventricular dissociation is a serious one. More than one-third of the patients considered are dead, with an average duration of six and half years after the onset of symptoms. The possibility of reversion to a normal rhythm is remote and would seem to occur only in those toxic or syphilitic conditions which are seen early and treated adequately. The age, the ætiology, the type of dissociation and the presence or otherwise of the Stokes-Adams syndrome are the important factors to be considered in all cases.

In the majority of cases the development of an auriculo-ventricular dissociation leads to a considerable reduction in the activities of the patient. The prognosis as to life would appear to vary in the different types already mentioned, being worse in those conditions associated with variable block and better in those associated with constant block.

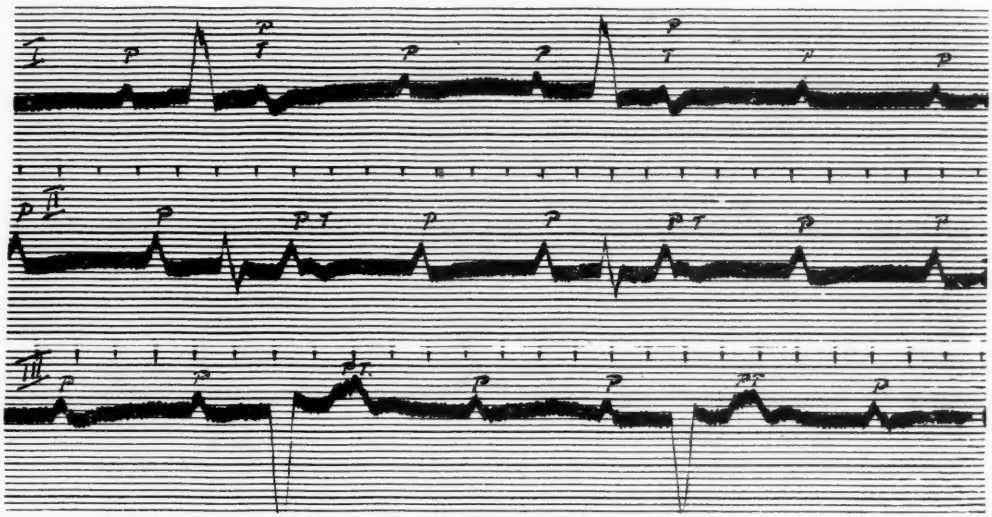

FIGURE XIV (Case X), October 18, 1928.
Showing a typical complete dissociation with superadded block in the right branch of the bundle of His.

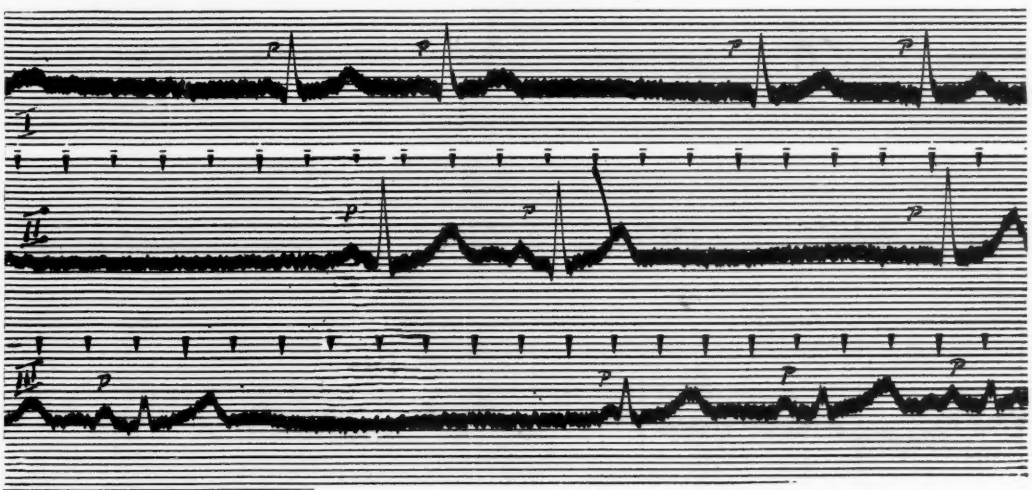

FIGURE XV (Case XV), June 2, 1928.
Showing a sino-auricular block. This patient has shown short attacks of syncope of short duration. No suggestion of auriculo-ventricular dissociation has been shown on any electrocardiogram from this patient.

Finally, the liability of all these patients to sudden death must be remembered and a guarded prognosis would appear advisable at all times.

Stokes-Adams Syndrome Occurring Apart from Auriculo-ventricular Dissociation.

The following two cases have not been included in the series so far considered, but are described here from the point of relevant interest.

CASE XV.—A male, *ætatis* sixty-one years, suffered from scarlet fever and diphtheria as a child. He came under observation in 1927, complaining of dyspnœa and præcordial pain on exertion; he had noticed symptoms for some months and had giddy attacks at intervals. He also had had several syncopal attacks in which he had lost consciousness for some seconds. On examination a slow irregular rhythm with a pulse rate of 42 was noted. His systolic blood pressure was 160 millimetres of mercury and his diastolic pressure 100 millimetres. An electrocardiographic record (see Figure XV) showed a variable degree of sino-auricular block with varying lengthy pauses of cardiac inactivity. This condition has remained unchanged and he has had one syncopal attack within the past six months. His ventricular rate rises on exercise and is slowed by ocular pressure.

CASE XII.—A female, *ætatis* fifty years, suffered from two attacks of rheumatic fever in childhood. She came under observation in 1926, complaining of palpitation and paroxysms of rapid heart action at intervals for fifteen years. Within the two years just prior to admission to hospital she had suffered several attacks of syncope and in one she was unconscious for half an hour, but had no convulsions. While under observation she had paroxysms of tachycardia in which the ventricular rate reached 240 a minute and the pulse was for the time imperceptible at

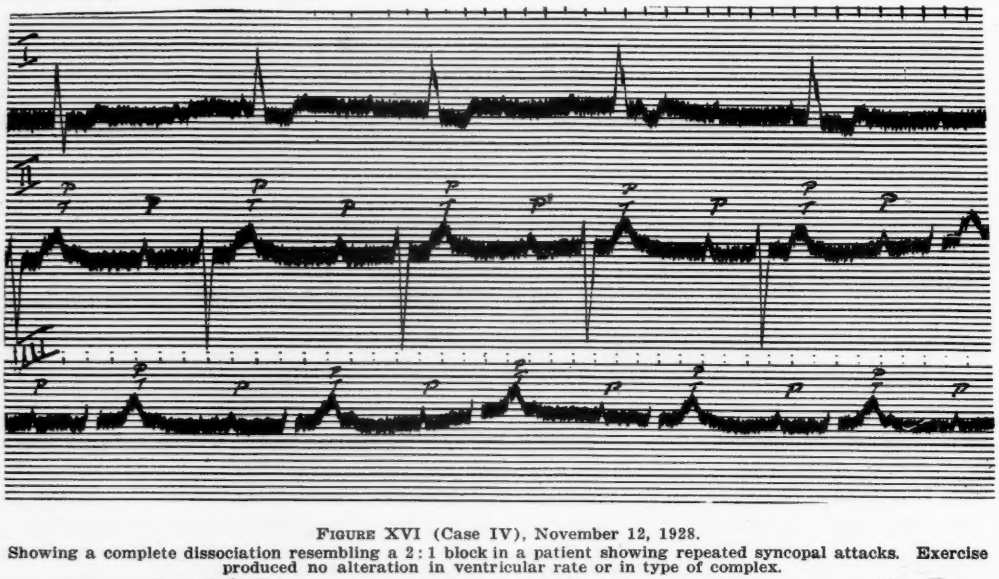

FIGURE XVI (Case IV), November 12, 1928.
Showing a complete dissociation resembling a 2 : 1 block in a patient showing repeated syncopal attacks. Exercise produced no alteration in ventricular rate or in type of complex.

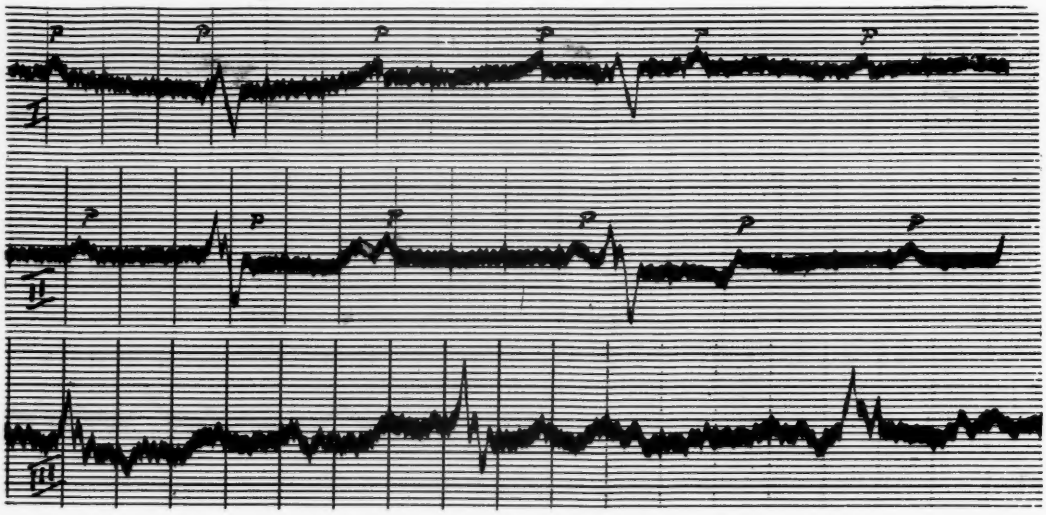

FIGURE XVII (Case XIV), October 13, 1927.
Showing a complete auriculo-ventricular dissociation with an apparent block in the left branch of the bundle of His.

the wrist. No syncopal attacks occurred during her stay in hospital. No effect was produced upon the tachycardia by vagal pressure.

I would submit that these two patients should be considered as manifesting the Stokes-Adams syndrome.

Conclusions.

A very close relationship would seem to exist between auriculo-ventricular dissociation and the Stokes-Adams syndrome. I have endeavoured to discover certain distinctions between those patients in whom the syndrome appears, and those in whom it does not. Only one distinction can be made which is that Stokes-Adams syndrome is comparatively uncommon in those who have a constant dissociation, while it is almost invariable in those who have a variable dissociation.

The proportionately higher incidence of the syndrome in younger people is probably due in part to difference in ætiology. I would suggest that, generally speaking, the causative factor is different in younger people and that the pathological process is of an active nature, affecting the bundle by impairing its function rather than destroying it and tending to produce a variability in block which may produce fatal syncope, but which also may appear to clear up completely on rare occasions. Whereas in older persons, frequently the subjects of myocardial damage, the pathological process is of a slower and probably sclerotic type, tending to destroy the bundle or so to affect its vascular supply that when once dissociation occurs, it remains constant.

Certain generalizations have already been made as to the prognosis of these various types of case.

The importance of influenza as a causative agent in the production of heart block would seem apparent, more particularly in younger people.

I am aware that the series is not a comprehensive one and I regret I have been unable to include cases of dissociation occurring following digitalis medication or in the course of acute specific fevers.

Finally, the cases have been worked out so as to present a clinical study of the condition commonly known as heart block.

Few collected series of cases of heart block have appeared in recent years. Strickland Goodall[44] has published a group of twenty cases and Willius[45] a group of twenty-two cases. There is a general agreement between these two series and the present one.

The rôle of syphilis as a causative agent much emphasized by earlier writers would seem of minor importance. One case occurred in Strickland Goodall's series, none in Willius's series and four in the present series. This view has also recently been expressed by Bickel.[46]

The high pulse pressure observed in these patients is described by Willius and has been confirmed by Norris, Bazett and McMillan.[47]

Treatment.

I propose to give a brief outline of the treatment, in order to illustrate the principles involved in dealing with this type of case.

The ideal aim is to abolish the dissociation which is present, but this will rarely be possible and only in cases of toxic or syphilitic origin. The removal of any toxic agent or focus, the treatment of acute infective diseases, such as influenza, along general

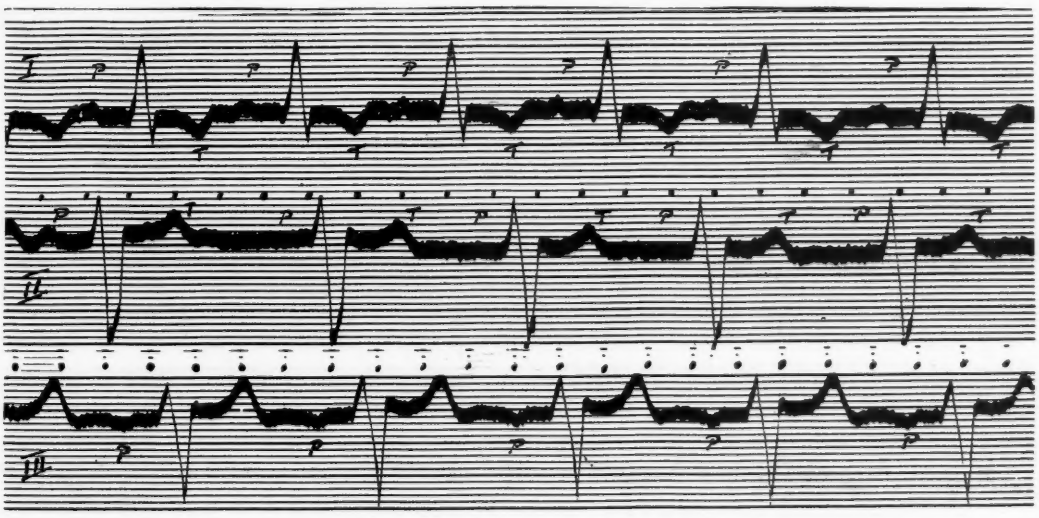

FIGURE XVI.I (Case XIV), November 12, 1928.

Showing no dissociation and a definite right branch lesion. I am unable to offer any adequate explanation of the sequence shown in these two plates. No electrocardiogram has been obtained in the interval and the patient's condition has improved. It is possible that a toxic and degenerative factor combined produced the original condition and that under treatment the toxic element has been removed, leaving a right branch block as a degenerative manifestation.

lines with ample rest in bed offers the greatest chance of reversion to normal rhythm.

In syphilitic cases the free inunction of mercury, combined with large doses of iodides by mouth is the ideal treatment. The enthusiastic intravenous administration of arsenic is to be condemned, as Bickel[48] has shown.

In those cases which are in the vast majority, in which the dissociation cannot be relieved, attention must be directed to the relief of symptoms.

The relief of cardiac embarrassment by treatment of cardiac failure, advocated by Levine,[49] is of importance. The nutrition of the heart should be considered in every case and may be improved by the liberal administration of sugar by mouth and by small doses of "Diuretin."

Activation of the central nervous system by aid of *liquor strychninæ* in a mixture is of value. It is a significant fact that these patients feel a considerable subjective improvement while taking this drug.

In some cases it is advisable to activate the ventricle by raising the irritability of the ventricular myocardium by barium chloride by mouth. Good results have been reported by Cohn[50] and Strauss.[27]

The syncopal attacks are best treated by subcutaneous injection of adrenalin, which in addition to activating the sympathetic system generally and raising the blood pressure, has a powerful action on the neuro-muscular mechanism of the ventricle as shown by Hume.[51] Phear and Parkinson[52] and other writers[53] [54] [55] [18] have reported successes.

Of late, ephedrine has been shown by Stecker[56] to be of value and it possesses the value of easier administration than adrenalin.

Dramatic effects are reported by Levine[31] of recovery after prolonged ventricular asystole by direct injection of adrenalin into the ventricle.

Finally, the liability of all such patients to sudden death must never be forgotten. A fatal issue may follow undue exertion or severe emotion and patients must be warned and guarded therefrom. Likewise, the embarrassment of the action of an already inefficient heart by an overdistended stomach is a real factor. It is of fundamental importance in all cases to guard against this by advising small dry meals with all fluids taken between meals. The importance of diet and avoidance of foods which tend to ferment and of fried and greasy food which delays digestion must be emphasized.

Lastly, the avoidance of intestinal stasis and constipation by periodic aperients should be remembered.

SUMMARY.

1. A brief account of the history of the Stokes-Adams syndrome is presented.

2. The recent literature on the subject is discussed.

3. The varying forms of the symptom complex are considered and an attempt is made to enunciate a comprehensive definition thereof.

4. A group of twenty-seven cases of auriculo-ventricular dissociation, including thirteen showing Stokes-Adams syndrome, is presented. A clinical

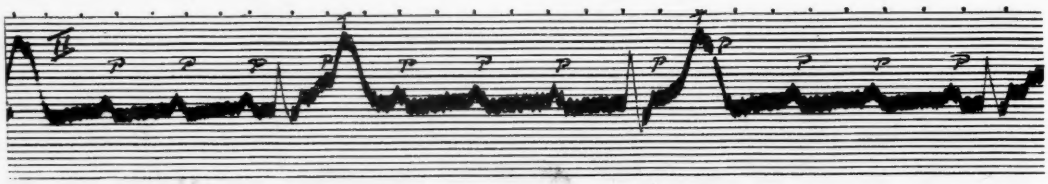

FIGURE XIX (Case XIII), January 30, 1926.
Showing complete dissociation with very large *T* waves.

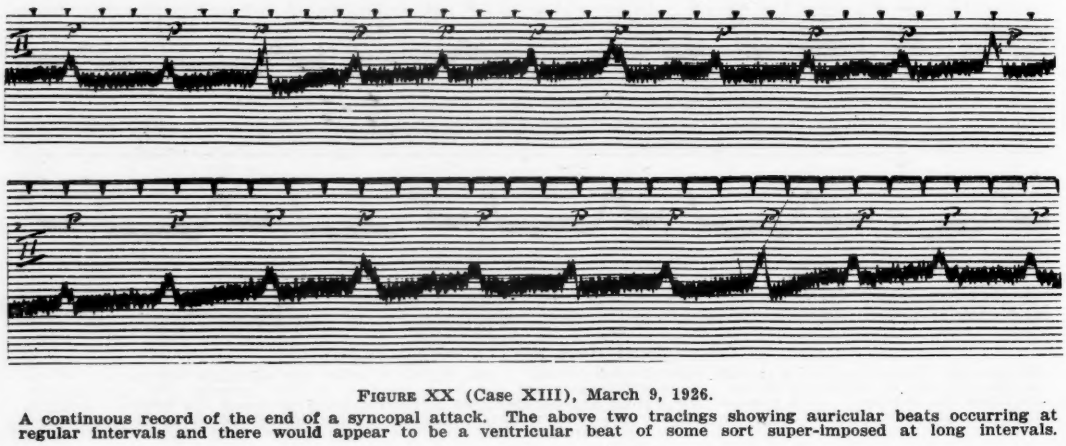

FIGURE XX (Case XIII), March 9, 1926.
A continuous record of the end of a syncopal attack. The above two tracings showing auricular beats occurring at regular intervals and there would appear to be a ventricular beat of some sort super-imposed at long intervals.

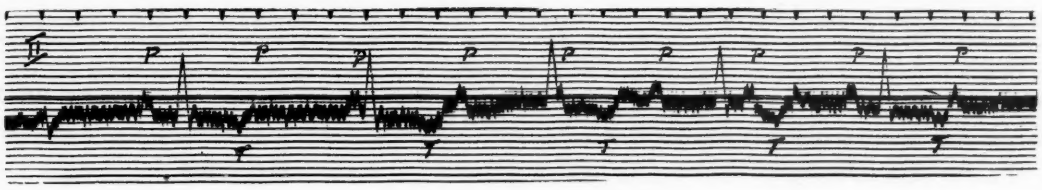

FIGURE XXI (Case XIII).

This continues from the above showing re-establishment of a ventricular beat with a totally different type of ventricular complex and persistence of complete dissociation.

FIGURE XXII (Case XIII).

Another record taken shortly afterwards showing another entirely different type of ventricular complex.

study is made and the close relationship of the two conditions is shown.

5. Two further cases manifesting Stokes-Adams syndrome from some other cause are described.

6. An outline of the principles of treatment is given.

ACKNOWLEDGEMENTS.

I desire to express my very sincere thanks to Dr. J. Strickland Goodall, who has not only placed his records and his patients at my disposal, but has stimulated my interest in cardiology by his example and teaching.

I express my thanks to Dr. F. J. Bach for the facilities he afforded me for the examination of patients.

The accompanying electrocardiograms have been prepared for me by Mr. H. W. Stowe.

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Reviews.

INFLAMMATION OF THE KIDNEY.

IN this small volume of one hundred and fifty pages written in simple language and entitled, "On Nephritis," Dr. A. C. Alport has amply achieved his aim "to produce a comprehensive but concise review of every phase of the disease within the covers of a relatively small book."

After a short historical summary of nephritis there follows a classification of the various types and although this is not so simple as Volhard's and Farr's classification, nevertheless it is possibly more complete since it includes such conditions as the Rose Bradford kidney *et cetera*.

In his description of these varieties the author endeavours to correlate chemical with laboratory findings without which it is not possible to arrive at a correct understanding of nephritis.

The chapter on chemical examination of the urine contains a very complete account of all the most useful renal function tests which are described in so simple a manner that the busy practitioner cannot read them without completely understanding the subject.

Analytical Department.

"DIABETONIT."

IN a prospectus of a preparation called "Diabetonit," manufactured by F. P. Winkler and Company, of Hamburg, it is stated that this substance is "an absolutely effective remedy for diabetes mellitus" and that it is "an absolutely harmless natural composition, in no way injurious to the body and made according to a special process. If used regularly every day, success is sure." It is described as a happy combination of antidiabetic ingredients, partly vegetable and partly containing nutritive salts.

At the request of the distributing agents we have submitted a sample to a competent clinician who has exhibited it strictly according to the directions given in the pamphlet. The patient was a man, aged twenty-nine years. He was suffering from severe diabetes, but his urine was free of sugar while receiving seventy units of "Insulin" daily. The patient offered himself for trial with "Diabetonit." On the day when the treatment was commenced his blood sugar was 0·27 milligrammes per hundred cubic centimetres. He was permitted to eat what he pleased and the "Insulin" was withdrawn. Sugar reappeared on the second day and five days after the first dose of "Diabetonit" he was acutely ill, on the verge of coma. The sugar content of the blood was 0·43 and the urine contained much sugar and acetone bodies. He was admitted to hospital without delay and within a very short time he became comatose. His condition during the following three days was desperate. With the help of "Insulin" he recovered and after a time his condition was the same as it had been before the experiment. It should be mentioned that the alleged remedy is recommended for "very serious cases." For patients in this condition one teaspoonful is to be given three times a day. "In these cases the doctor's advice may be asked." The physician refused to give it to another patient with severe diabetes and in this determination we concur. It is obvious that this secret preparation is quite useless and may be very dangerous. We warn members of the medical profession not to be misled by the untrue statements printed in the pamphlet.

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[1] "On Nephritis (for the Student and Practitioner)," by A. Cecil Alport, M.D. (Edin.), M.R.C.P. (London), with an Introduction by Professor Frederick Langmead, M.D., F.R.C.P.; 1929. William Heinemann (Medical Books) Limited. Crown 8vo., pp. 190. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 22, 1929.

The Making of Specialists.

IN the March issue of *The Journal of the College of Surgeons of Australasia* information was published in regard to the creation of new positions at the Alfred Hospital, Melbourne. The new positions are those of emergency surgical officer and clinical assistant to indoor surgeons. Emergency surgical officers are to be called upon to undertake the immediate treatment with operation, if necessary, of those emergency conditions for which the services of no in-patient or out-patient surgeon are available. Clinical assistants to indoor surgeons are to be attached to one in-patient surgeon, are to attend on that surgeon's operating day or days and are to perform such duties as the surgeon may direct. Clinical assistants are required to furnish evidence that they have taken or are taking special pains to improve their knowledge and technique of surgery. It will be seen at once that the emergency officer will necessarily be a surgeon of some experience. The clinical assistant may or may not have had adequate training as a surgeon, but he would have a wonderful opportunity of improving his "knowledge and technique of surgery." This is a reversion to the old system of apprenticeship and is undoubtedly the best way of securing efficiency. It is quite unnecessary to point out that surgery means more than skill in the performance of operations. To the theoretical basis of surgery, the word being used in its widest sense to include anatomy and a knowledge of the clinical and pathological manifestations of disease, must be added judgement born of experience. The fact that the appointee was taking special pains to "improve his knowledge and technique of surgery" would satisfy the theoretical requirements and the close association with an experienced surgeon would do more than anything else to lay the foundation of those habits of caution and method that would eventually justify the title of surgeon.

Since this method of progression is so valuable in the training of surgeons, there is no doubt that it can be applied to the study of general medicine or of diseases of special systems. Medical practitioners in Australia who desire to become proficient in the practice of a certain specialty, labour under several disadvantages. Post-graduate courses of instruction are held in several cities of the Commonwealth every year. These are of short duration and, excellent though they are, they are with few exceptions not arranged so that anyone desirous of specializing in any branch of medicine can use them as a basis for his work. The best that a would-be post-graduate student can do is to attend the out-patient and in-patient clinics at the teaching hospitals and to rely on the good graces of the honorary officer to point out features of interest. He will certainly see what is going on, but he will have no opportunity of receiving systematic instruction and no opportunity of doing any work himself in either investigation, diagnosis or treatment. He knows that if he goes to Great Britain or the Continent it will be possible for him to make more satisfactory arrangements. Here he will be permitted to work in the clinic of one man, he will be that man's pupil, he will be taught much of the theoretical side of his subject and, if he is studying a subject in which laboratory work or operative work is essential, he will be allowed to do the actual work himself under proper supervision. Australia should be able to provide facilities such as this for the serious student. It should be possible to arrange for the appointment of clinical assistants in such a way that some of the difficulties would be overcome.

The first step in the arrangement of such a scheme would be to secure the cooperation of the boards of management of the metropolitan teaching hospitals. Arrangements might be made for the payment of a certain fee or premium to the hospital by the clinical assistant. The board would appoint him to be clinical assistant to one honorary physician, surgeon or specialist for a period of six or twelve months. The appointment would have to be made on the

recommendation of the honorary medical officer, for if the latter is to accept a pupil for a definite term, he should have a voice in the appointment. Both the honorary medical officer and the board would require evidence that the candidate for appointment was a fit and proper person to receive instruction and that he had already engaged in the study of the subject to such an extent that he had sufficient knowledge to justify the appointment. After appointment the clinical assistant would be in constant attendance on his teacher, he would receive instruction in the examination of patients and in diagnostic methods, he would follow the treatment through all its stages and would perform certain operative procedures under the personal supervision of the teacher. The performance of surgical operations, when these were required, would be necessary. A man may see a dozen or more operations, such, for example, as submucous resection of the nasal septum or radical cure of antral disease, but unless he performs one or two under the supervision of a senior, he will not feel competent to undertake the procedure alone when occasion arises. The honorary medical officer should not receive a portion of the premium paid by the trainee; the honour of being regarded as a post-graduate teacher and the status attached to the work ought to be sufficient recompense. The fee paid to the hospital should be adequate. The hospital authorities would find that the quality of the work carried on within its walls would not suffer in the least degree. On the contrary, with more intensive teaching the standard of work always reaches a higher level. There is one point in conclusion which needs emphasis and that is the fact that six months' apprenticeship in the study of a specialty will not make a specialist. It would give a man a ground work on which he could build. The character of the edifice would depend largely on himself. A scheme of such a nature as that outlined merits the serious consideration of the authorities of the teaching hospitals. It is a matter in which the first move might be made either by the medical faculties of the several universities or by some body such as the Melbourne Permanent Committee for Post-Graduate Work.

Current Comment.

CYTOTOXINS.

IN the endeavour to determine the nature of cells comprising malignant growths innumerable experiments have been carried out having for their object the production of immunity to certain tumours. It is generally assumed that in spontaneous tumours the cells start as normal tissue cells and the limitless overgrowth is an acquired character which distinguishes the malignant from the normal cells. Under rare conditions malignant tumours have been observed to degenerate and to disappear. It has been suggested that in these circumstances the tissue fluids have acquired the power to disintegrate the component cells; in other words, a cytotoxin has been produced. It is, however, possible that normal tissue cells are prevented from being destroyed by antibodies by a special protective mechanism and that this protection continues to reside in tissue cells when they form malignant tumours. It will be recognized that the study of cytotoxins and of protective substances is surrounded by many technical difficulties and that these difficulties are multiplied when it becomes necessary to seek information through indirect methods. Bashford, Ehrlich, Lewin and others succeeded many years ago in establishing a definite immunity to certain forms of carcinoma in the mouse by injecting embryonic skin and other epithelial tissue. The immunity was determined by the consistent failure to induce mammary mouse tumours by inoculation. This form of immunity is now well recognized, although opinions still differ concerning the actual mechanism of the process. The immunized mouse does not seem to be harmed by the immunization. Its normal epithelial structure remains untouched; growth is not impaired and the only ascertainable change appears to be that it is no longer possible to transplant a small mass of mammary carcinoma from another mouse. If the immunity is dependent on a cytotoxin, this must be specific for malignant cells and not active to the original epithelial cells of the growth.

The injection of tissue cells of one species into an animal of another species gives rise to the production of a precipitin. The first result is an antibody active to protein generally; the later result is an antibody specific to the protein of the animal whose cells were used for the immunization. There is no evidence at present to suggest that this protein reaction is of any significance in the problem of malignant growths. While it is possible to graft a tumour from a mouse on to a rat or other animal, spontaneous tumours arise from the cells of the animal's own tissues and therefore no heterologous antibody can be formed.

Some highly interesting experiments have been carried out at the Lister Institute by T. Lumsden and A. C. Kohn-Speyer in connexion with the nature of cytotoxins.[1] Unfortunately these investigators

[1] *The Journal of Pathology and Bacteriology*, April, 1929.

have adopted a method of approach that is so complicated that deductions from their findings have to be subjected to the strictest scrutiny. They found in their earlier work that the normal serum of an animal does not damage artificial cultures of cells of the same animal, but is toxic to the cultures of cells from animals of another species. They have endeavoured to study the mechanism of this alleged cytotoxin. They found that when normal mouse cells, except spermatozoa, or mouse cancer cells were cultivated *in vitro*, no effect was produced by the addition of normal mouse serum, some lethal effect was produced by normal rat serum and a powerful effect was produced by serum of an animal far removed in the evolutional scale. If the toxic normal serum be heated to 56° C. for one or two hours or kept without heating for one or two days, it loses its toxicity. The serum was found to be toxic to spermatozoa of the same species. This spermotoxic action was demonstrated with normal rat's serum and with the serum of a rat immunized against rat testes. But the normal rat serum lost its toxicity after standing for two days, while the serum of the immunized rat remained toxic. Lumsden holds that normal serum contains a cytotoxin ready to repel any invading foreign cell. If this defence fails, as it is not as a rule a powerful one, the serum acquires a second force as a result of contact with the formed elements of the blood.

In the next place Lumsden found that when a mouse tumour was grafted on to a rat and had grown for over one week, the serum of the rat exercised no damaging action on cultures of the mouse tumour cells, although it proved to be toxic to cultures of the same tumour cells that had not been grown in rats. Similarly he found that if a mouse or rat malignant tumour is used to immunize a rat, its serum is toxic to artificial cultures of tumour cells derived from a mouse, whether the tumours be originally mouse or rat tumours, but is indifferent to cultures of the cells of either tumour, provided that the cultures were made from tumours growing in a rat. The same applied to the serum of a mouse immunized against one or other tumour. He interprets these results by the assumption that when a mouse tumour cell gains protection against rat-made antibodies, it loses its original protection against mouse antibodies. This assumption cannot be accepted without more convincing evidence. In the first place it is questionable whether the arrest of growth of tissue or tumour cells *in vitro* can be ascribed to definite antibody action, partly because the growing cells are extremely delicate and difficult to cultivate and partly because the fact that the tumour of a mouse can be grown in a rat proves that a material cytotoxin to rat tumour cells does not exist in a normal mouse. The phenomenon appears first when the cells are induced to grow outside the animal body. The first objection is upheld by some experiments undertaken by F. C. Pybus and H. R. Whitehead.[1] These observers had difficulty with the culture medium for the artificial growth of tumour cells. They found that when they used equal volumes of mouse serum and Ringer's solution, with or without embryo extract, the serum of a rabbit immunized to mouse cancer was toxic to cultures of the cancer cells and also to cultures of heart and kidney cells. In other words, they claim that Lumsden's technique had led to the appearance of a tissue specificity which could not be demonstrated when the mouse cancer cells were grown in a more dilute medium. They therefore are unable to agree that there are antibodies specific to the different tissues of the animal body. They argue from this that it is obvious that there cannot be any antibodies specific to malignant cells and not to normal tissue. As far as the phenomena demonstrated are concerned, it appears that normal serum of one animal is destructive to cells of another species growing *in vitro* in a serum medium, but not to the cells of the same species of animal. The conditions governing artificial culture of cells, however, are not fully known. Under the most favourable circumstances the growth is not like that which occurs in an animal body. It would therefore be hazardous to accept findings that are secured under one set of experiments and not under another, unless these findings harmonize with observations made in the animal body. Pybus and Whitehead have further endeavoured to ascertain whether antibodies exist that are common to malignant cells of animals of different species. They immunized rabbits with human carcinoma cells and tested the serum of these rabbits against mouse carcinoma, heart and kidney cells. It was found that although the growth of these cells was less good in the presence of immune rabbit serum than in the presence of normal rabbit serum, it was still quite free in the former. They are not disposed to draw any conclusions from the results. We are therefore left with the knowledge that tissue culture in its present form is incapable of enabling the cytologist to differentiate the serum of an animal with malignant disease from that of an animal not so affected.

We are still ignorant concerning the mechanism that safeguards the normal cells of an animal body against destruction. Whatever that protection may be, it is probable that it extends to the cells of a malignant growth, that is to normal cells that have taken on limitless growth and have ceased to fulfil the physiological obligations of their ancestors.

[1] *The Journal of Pathology and Bacteriology*, April, 1929.

THE PROFESSOR OF OBSTETRICS IN MELBOURNE.

THE Council of the University of Melbourne has adopted the recommendation of the special committee and of the Faculty of Medicine and has appointed Dr. R. Marshall Allan, M.C., Professor of Obstetrics. The history of the movement leading to this appointment has been published in this journal from time to time. The new professor will devote the whole of the time to teaching and research. We offer him our sincere congratulations.

Abstracts from Current Medical Literature.

MEDICINE.

Ulcerative Colitis.

J. A. BARGEN (*The Journal of the American Medical Association*, October 20, 1928) discusses the cause and symptoms of chronic ulcerative colitis. The ulceration begins in the rectum usually and spreads upwards to involve the whole colon. Frequent passages of blood, mucus and pus with tenesmus are the rule; slight to serious fever occurs and anæmia with debility is common. A moderate polymorphonuclear leucocytosis occurs. A diplostreptococcus with definite cultural, biological and morphological properties has been isolated from the stools in pure culture in 189 cases. Injected into the veins of rabbits these strains caused diarrhœa, hæmorrhages or ulceration in 268 out of 459 healthy rabbits. The organism was isolated from the blood in five instances. Predisposing factors are upper respiratory infections, dental and tonsillar sepsis, overwork, lowered resistance and other bowel infections. Proctoscopic examination reveals a typical picture of myriads of miliary abscesses and ulcers with inflamed areas between. Later X rays reveal a narrowed, non-haustrated colon. The mucosa eventually becomes denuded from almost the whole colon. Complications include malignant disease, polyposis, perforation, stricture, hæmorrhage, endocarditis, splenomegaly, tetany and arthritis. Treatment includes the use of an autogenous vaccine or a specific immune serum in acute cases; removal of all foci of infection and a liquid diet in acute cases increasing to a high calory, high vitamin and low residue diet in chronic cases. Fresh air, sunshine, mental diversion and encouragement are necessary to overcome the mental distress. Rest in bed should be abandoned early. Bismuth, kaolin and opium may help the diarrhœa. "Acetarsone" by mouth may be beneficial. Irrigation of the colon is disappointing in its results. Surgery is rarely needed, though ileostomy may save life if the condition is progressive. Caecostomy and ileo-sigmoidostomy are rarely helpful. Six hundred and fifty-five cases were analysed; ninety-three patients died. The results of vaccine therapy were on the whole good; 69% of patients were restored to ordinary usefulness.

Bright's Disease.

T. ADDIS (*American Journal of the Medical Sciences*, November, 1928) discusses the renal lesion in Bright's disease. Clinically Bright's disease is a condition in which albumin, casts and red cells appear in the urine in abnormal quantity. By using an acid alcoholic solution of phosphotungstic acid the amount of albumin in normal urine was estimated and it was found that the normal person excreted up to thirty milligrammes of protein in twelve hours. There are many conditions of the urine in which casts are dissolved, but it was found that casts and red cells could be demonstrated most readily in concentrated acid urine. Thus if a person abstains from fluid during the day and collects the night urine for a twelve-hour period, casts and red cells can be found most readily, except when a large part of the kidney is destroyed by disease. In such circumstances the kidney cannot excrete urea sufficiently, the blood urea rises and dilute urine is excreted; casts and red cells are then not readily observed. It was found that normal subjects excreted up to 5,000 casts and up to 425,000 red cells in a twelve-hour period. The average red cell excretion in seventy students was 67,000 in twelve hours. Working on this basis it was possible to state how far the excretion of protein, casts and red cells in any urine differed from the normal. In this way three varieties of chronic Bright's disease were separated. The first was the arterio-sclerotic in which the blood urea to urine urea ratio was altered, with increase of blood urea, but in which the output of protein casts and red cells was only slightly above normal. The second was the degenerative in which casts and epithelial cells were much increased, and the third was the hæmorrhagic in which red cells occurred in considerable excess. It was recognized that a degenerative or hæmorrhagic lesion can be superimposed on an arterio-sclerotic one. In seventy-five instances *post mortem* examinations revealed in the arterio-sclerotic group renal arterio-sclerosis and a patchy fibrosis of the cortex; in the degenerative type granular, fatty and necrotic changes in the tubule cells and in the hæmorrhagic group inflammatory lesions in the glomeruli.

Human Infection with the Bacillus of Bang.

F. WEIGMANN (*Klinische Wochenschrift*, February 19, 1929) states that in agglutination tests performed on 1,009 patients in Schleswig-Holstein a positive result with the bacillus of Bang was noted in thirty-two instances. The sera reacting to the bacillus of Bang did not give positive results with either typhoid or paratyphoid cultures. In six of the cases direct infection from animals was noted. In nineteen there was sufficient proof that the disease had been contracted by drinking raw milk from infected animals. Although in his series there were no instances of infection through the skin, he believes that this also plays an important part. The great majority occurred in males and there were no infections in children under twelve. The main symptom is fever of the remittent or intermittent type often exceeding 40° C. (104° F.). The general condition of the patient and the low pulse rate are in sharp contrast with the undulant temperature curve. More chronic infections may cause some splenic enlargement and the blood picture is one of leucopenia with relative increase in lymphocytes. The disease may continue for months, although in many instances the possibility of fresh infection must be considered.

Complement Fixation Test in Gonorrhœa.

W. SCHULTZ AND J. DÖRFFEL (*Deutsche Medizinische Wochenschrift*, January 25, 1929) have made exhaustive experiments on the value of the complement fixation test, especially as regards the diagnosis and cure of gonorrhœa. They conclude that it is specific for gonorrhœal diseases and of considerable diagnostic importance. In acute infections, except in patients receiving immediate treatment, the result was positive in 75% and in 80% of those whose infection was chronic or those with complications. In a small number the result was positive during the first two weeks, but in the majority the reaction did not develope until the end of the fourth week. In a limited number it did not appear for nearly three months. In many chronic infections with and without complications the result remained positive for long periods up to one year, although no organisms could be detected. Provided a positive response subsequently fails to be manifest, a certificate of cure can be safely given, but if the reaction continues in the absence of local signs of infection, the authors are uncertain whether it should be accepted as proof of possible infectivity. As with the Wassermann test, they emphasize the value of using several antigens in the performance of the test.

Bilateral Peripheral Facial Paralysis.

S. FRANKE (*Münchener Medizinische Wochenschrift*, March 1, 1929) describes a case of bilateral peripheral facial paralysis due to syphilis. The paralysis was noted five days after the onset of the cutaneous eruption and cleared up after five injections of "Bismosalvan." Facial diplegia of rheumatic origin is fairly common, but such a nerve injury of syphilitic origin is rare. Other causes of bilateral palsy are thrombosis of the basal artery, meningitis, trauma or tumour formation of the base of the brain as well as inflammatory conditions of the Fallopian canal. Some authorities consider such syphilitic manifestations to be due to a premature tertiary lesion, while others believe it to resemble a toxic neuritis such as follows lead, alcohol or diphtheritic toxins.

Pernicious Anæmia.

L. VON VARGA (*Deutsche Medizinische Wochenschrift*, March 15, 1929) gives the details of eight cases of pernicious anæmia in which "Insulin" alone or in combination with liver extract gave good results. "Insulin" was first tried in a patient with a severe anæmia with signs of coma. Within a week a great improvement

was noted with return of colour, restoration of appetite and rapid improvement in the blood picture. In general appetite returns first, tinnitus, giddiness and debility diminish, but headaches take longer to disappear. The lips and cheeks begin to colour in about ten days and rapidly improve. The increase in red corpuscles and leucocytes is pronounced, although the colour index remains unaltered for a considerable period. The only symptom unaffected is achlorhydria. As the literature contains few references to the use of "Insulin" alone or combined, these results are claimed by the author to be a distinct advance in current therapy.

Endarteritis Obliterans.

C. E. BENDA (*Deutsche Medizinische Wochenschrift*, March 15, 1929) describes a case of *endarteritis obliterans* in a man, aged thirty-two years, who had previously been in good health. After two weeks of severe pains in the legs the symptoms of intermittent claudication developed. Later gangrene of one small toe occurred; the toe was treated by amputation. With complete rest and medical treatment the progress of the disease was arrested and slow recovery took place. Syphilis plays an important part in the ætiology of the condition, although in this instance, despite repeated tests, no reaction was obtained to the Wassermann test. Other toxic substances such as tobacco, have been blamed on rather unconvincing evidence. Any existing basal cause, such as syphilis, gout, diabetes, must be treated. Iodine is useful, but not in the large doses usually given. The author recommends a mixture of sodium iodide and sodium bromide, 0·2 gramme of each in 200 cubic centimetres of water. Aspirin in doses of not more than 0·5 gramme acts as a sedative. Cardiac tonics may be required when strophanthus is used and not digitalis. Local treatment by hot baths and diathermy is useful in the later stages and complete rest in bed is essential. Provided that the diagnosis be correct, the prognosis is not unfavourable following on careful treatment.

Diabetes.

A. W. KAPLAN AND J. KONOPNICKI (*La Presse Médicale*, October 3, 1928) describe their methods of treating diabetic patients with "Insulin." "Insulin" should be used only in those patients in whom diet alone does not improve the carbohydrate metabolism and overcome acetonæmia. Such patients fall into two groups, one in which pronounced glycosuria and acetonuria are corrected by diet and small doses of "Insulin" with apparent improvement in tolerance and the other in which large doses of "Insulin" are required, but the tolerance gradually declines. In the first group some islets of Langerhans remain and function better after treatment; possibly there is some regeneration. In the second group the pancreatic degeneration tends to increase. A restricted diet, twenty-five to thirty-five calories per kilogram of body weight, is beneficial, even with "Insulin" as a rule, but in some severe diabetes the metabolism of fats and proteins is disordered and glycosuria and acetonuria persist in spite of "Insulin"; in these cases greater amounts of carbohydrate and "Insulin" may bring about an adjustment. It is thought that this is due to an increased storage of glycogen in the liver, because the fats and proteins require the presence of a certain amount of glycogen in the liver for their combustion and, if this falls below a certain point, it can be replenished only by introducing abundant carbohydrates rendered assimilable by "Insulin." In any case the diabetic subject should be given the full amount of carbohydrate which he can tolerate. No benefit is to be derived from restricting the intake below that figure. It is as if the burning up of the full amount of carbohydrate was beneficial by stimulating the pancreas. Fasting days are useful in mild diabetes, in fat people or in those not emaciated; in severe diabetes any benefit is temporary and often followed by an increase in the amount of sugar. The usual *régime* for diabetics on admission is a standard diet containing 48 grammes of carbohydrates (without counting vegetables), 50 grammes of protein, 130 grammes of fat, about 1,600 calories; 500 to 1,000 grammes of green vegetables cooked in three waters are allowed. This method reduces the content of carbohydrate to about 2·5%. The remainder of the diet consists of potatoes 200 grammes, eggs seven, cream 180 grammes, butter 100 grammes. Bouillon, coffee, tea or lemonade with saccharine are allowed in any quantity. An occasional fast day is advised. If after a week on this diet glycosuria is present, "Insulin" is used. In severe diabetes with definite acetonuria treatment begins with a diet of broth and large doses of "Insulin"; the broth is made up of 125 grammes of prepared oats (Herculo), 125 grammes of butter, served in five portions. This contains 90 grammes of carbohydrate, 20 grammes of protein, 100 grammes of fat. If this is not successful, thick soup is given containing 600 grammes of green vegetables, 200 grammes of peas and haricots, 100 grammes of butter, 180 grammes of cream. The vegetables used are much better tolerated than bread. This soup is effective in getting rid of acetone. When sugar tolerance has improved, a gradual return is made to the first mentioned diet. Eventually an endeavour is made to allow a diet of 100 grammes of carbohydrate, 100 grammes of protein and 150 grammes of fat, particularly for young working people; fast days or semifast days are interpolated. As a rule "Insulin" is given in doses of 20 to 80 units a day and later an attempt is made to reduce the dose by five units every second or third day, until the minimum dose is determined on which equilibrium is maintained. The use of very high doses does not increase the activity of "Insulin." Sometimes five to ten units a day are sufficient. As a rule "Insulin" is given twice a day, a larger dose at 7 a.m., a smaller in the evening. In severe diabetes three to five doses a day may be used with carbohydrates correspondingly distributed. As a rule the blood sugar rises in the night, is high in the morning and falls towards midday. In the aged, in hypertension, atheroma and heart failure, "Insulin" should be used with caution, as it may aggravate the general ill health. In hypertension and atheroma a high blood sugar is sometimes noted apart from diabetes. In such circumstances small doses of "Insulin" may be used. In coma only three patients out of six recovered in spite of large doses of "Insulin" and of glucose. In pulmonary tuberculosis "Insulin" often renders great service and surgical operations are made safe by its use in diabetes. Ulceration, severe pains and gangrene of the extremities yield to "Insulin" if they are not due to anatomical narrowing of the vessels (*endarteritis obliterans*); in the latter "Insulin" fails.

Occult Syphilis.

A. ANGUERA (*Boletin Tecnico de la Dirrecion General de Sanidad*, March, 1929) defines occult syphilis as the occurrence of the disease in patients who are ignorant of its presence, not as syphilis which escapes detection by the medical practitioner owing to lack of thorough investigation. Fournier fixed the percentage of occult syphilis as three in men and eighteen in women. He died, however, before the discovery of the *Treponema pallida* and the use of serum reactions came to the aid of the clinician. Later writers gave much higher figures. Anguera does not include in his series those patients who wilfully mislead the medical practitioner as to their venereal history and those who owing to the level of their mentality do not notice most salient facts affecting their health. He deals with those infections occurring in persons who are most careful of their health, and who discuss it most frankly with their medical advisers. These are they who, when convinced of their condition as a result of a serum reaction, declare in all sincerity that they have never noted any of the stigmata of syphilis on their bodies. He gives records of four patients. The first was a foundry worker whose chief complaint was a recurrent vertigo which caused him to lose his balance at times. He also complained of sore eyes, due, he supposed, to the effect of the heat and the bright glare from the furnaces. He was a brisk, healthy man with no signs of syphilis and had a healthy wife and four healthy sons. Antisyphilitic treatment brought about a cure. The second patient was a young French painter. His condition had been diagnosed as Raynaud's disease, but he gave a Wassermann reaction and antisyphilitic treatment brought

about a complete cure. The third patient was a married woman who had had a number of abortions. No trace of syphilis was found in herself or her husband. Her blood gave a positive response to the Wassermann test. After treatment she gave birth to a healthy child. A sister of this patient was also found to be syphilitic. The family history showed that both parents died of cerebral trouble. During later years they had ulcers of the legs which resisted treatment. Probably the daughters had inherited syphilis. The last case was that of a foreign lady of high social standing. She had been treated for rheumatism by leading doctors in Madrid, but had received only transitory benefit. In the course of a complete blood examination a Wassermann reaction was obtained. The results of flocculation tests were also positive. Intravenous injections of "Neo-salvarsan" caused a disappearance of her troubles. Anguera's investigations lead him to conclude that with present day methods occult syphilis is increasingly easy to diagnose. This is particularly true of those who make a special study of venereal disease, and consultants.

Climate in the Treatment of Pulmonary Tuberculosis.

J. A. MILLER (*Tubercle*, February, 1929) discusses the value of climate in the treatment of pulmonary tuberculosis. The factors which are of importance in assessing climate are temperature and humidity, wind and rainfall, the character of the soil and the configuration of the country, whether mountainous or flat. Temperature and humidity are closely interrelated, the effects of dryness being intensified by cold and those of moisture by heat. Whereas medical opinion once favoured equability in climate, it is now becoming generally recognized that a variable climate is to be preferred, in moderation. Sunlight has a definite physiological effect, but rain, if periodic and not excessive, is beneficial in enhancing the variability of climate. The author stresses the fact, however, that the regimen of regulated rest and exercise, of proper food and open air life is the essential basis in the treatment of tuberculosis. When these essentials are assured, a change of climate is of definite value in the majority of instances, but he considers that under strict supervision many patients will do well in any climate. No patient should be sent away in search of climate unless he can afford to stay away for a minimum of from six months to a year and to have the necessary food, lodging and medical care. The author strongly deprecates the practice of simply sending patients to the country or the mountains, with the advice that they do not bother about medicine or doctors. Every patient should be sent either to a well recognized institution or to a physician skilled in the management of tuberculosis. Only thus can the proper regimen be assured, especially in the regulation of rest and exercise, a most important factor, which no patient can carry out by himself. In acute tuberculosis with fever or hæmorrhage or when the condition is far advanced, absolute rest in bed is the essential and change of climate, involving the fatigue of travel is contraindicated. The choice of locality depends on a number of factors, including age, the presence of complications, evidence of cardio-vascular disease and the temperament and tastes of the individual patient. The cold, dry and variable climate of the mountains is suitable for those who are young or who have vigorous constitutions. Dry, sunny climates are preferable for those with laryngeal involvement or those with bronchitis. Equable, mild climates at low altitudes should be chosen for the elderly and for those of nervous temperament, as well as for those with arterio-sclerosis, weak hearts or a definite tendency to dyspnœa. From an analysis of his own cases, numbering over one thousand, the author concludes that good results may be obtained in any locality, provided the physician selects the particular locality according to a definite plan. Further, as he has obtained the same or even better results in those patients who have never left the environment of New York City, than in those who have been in distant health resorts, he draws the conclusion that climate *per se* is not the determining factor in the successful treatment of pulmonary tuberculosis.

Primary Carcinoma of the Lung.

C. V. WELLER (*Annals of Internal Medicine*, February, 1929) publishes the histories and autopsy findings in twelve cases of primary carcinoma of the lung. The diagnosis was confirmed by microscopical examination in every instance. The author stresses the necessity of constantly keeping in mind the possibility of the presence of this condition which is not so rare as is generally supposed. He states that primary carcinoma of the lungs and bronchi is found once in every two hundred autopsies upon adults in Europe and America and once in every twenty deaths from carcinoma. In making a positive diagnosis much help is gained from bronchoscopy for inspection and excision of a portion of the growth for microscopical examination and from X ray examination with the assistance of "Lipiodol" injections in selected instances. The signs and symptoms fall into four groups: (i) those of the local chest lesion (such as cough, sputum and effusion), (ii) those of mediastinal tumour (such as venous engorgement and œdema of the upper part of the body, inequality of the pupils and recurrent laryngeal paralysis), (iii) general systemic effects (such as cachexia and fever), (iv) signs and symptoms frequently associated with metastases (such as pain in the back and headache). In only four of the twelve instances was a correct diagnosis made during life. In one other instance the condition was thought to be intrathoracic malignant disease, probably lymphosarcoma, and in two instances abdominal malignant disease was diagnosed. In spite of the fact that treatment is usually of no avail in this condition, a correct diagnosis may be of great value to the patient and his relatives and prevent his unnecessary admission to a tuberculosis sanatorium.

Ætiology of Backache.

G. L. LAMBRIGHT (*Annals of Internal Medicine*, February, 1929) discusses the various causes of aches in the lumbar and sacral regions. He distinguishes three main groups, first, backache from systemic diseases, secondly, from local conditions and, thirdly, from reflex causes. The first group comprises acute and chronic infectious states and metabolic disorders, such as hypothyreoidism. The local conditions include sacro-iliac strain, anomalies of the vertebræ, spondylitis and spinal tumours. Among the reflex causes may be mentioned pelvic disease, prostatitis, renal calculi and sexual excess. The author stresses the necessity of a thorough physical examination, including inspection of the back, palpation of the sacro-iliac synchondroses, abdominal palpation and examination of the pelvis and rectum. Septic foci should be sought, especially in the teeth, tonsils, sinuses and prostate. The urine should be examined and in some instances pyelography is necessary. An X ray examination of the lumbar and sacral regions may give important information. Only such special examinations as are considered necessary after the physical examination, need be performed. No field of medicine requires greater skill in diagnosis. He concludes that the cause of backache is not more often discovered because of the lethargy of medical practitioners.

Immuno-Transfusion in Subacute Bacterial Endocarditis.

C. M. KUNTZ AND P. D. WHITE (*The New England Journal of Medicine*, March 7, 1929) in reporting the case of a patient suffering from subacute bacterial endocarditis who was treated by means of transfusion from immunized donors, briefly review the literature dealing with this method of treatment. The disease is usually fatal, however treated. The *Streptococcus viridans* is the causative organism in almost all cases. Dr. Hans Zinsser was consulted by the authors with regard to proper procedure of immunizing the donors. He advised that a series of six injections of the vaccine prepared from the culture of the patient's blood be given to the donor over a period of three weeks. The donor's serum was titrated for antibodies at the end of this period and it was found that the patient's serum contained a higher titre than did that of any of the donors. The following procedure was adopted in giving the transfusions. Before the first transfusion a litre of blood was withdrawn from the patient and he was then given one thousand eight hundred

cubic centimetres of immunized blood at one time, six hundred cubic centimetres from each of three donors. Five smaller transfusions each of five hundred cubic centimetres were given at various intervals, the last transfusion being given three months after the first. No definite change was noticed as a result of the treatment. The disease progressed unabated and terminated fatally about seventeen months after the onset of the illness. The treatment was instituted before there was any great degree of anæmia. It is noteworthy that the patient developed an active immunity which was greater than that induced in the donors by the injection of the vaccine.

The Cholesterol Content of Blood Plasma in Diabetes Mellitus.

I. M. RABINOWITCH (*Archives of Internal Medicine*, March, 1929) discusses the value of the estimation of the cholesterol content of the blood plasma in *diabetes mellitus*. Two thousand estimations were performed. The blood was obtained from the patients in the fasting state at least fifteen hours after the evening meal. Other writers, notably Joslin and Professor Bloor had shown that all types of *diabetes mellitus* are distinguished by a pronounced increase in the blood lipoids, the increase being progressive with the seriousness of the disease. It had also been demonstrated that there was no parallelism between the fluctuations in the blood sugar and in the blood lipoid. The author confirms and amplifies this work. He used the estimation of the plasma cholesterol as an index of the lipoid metabolism, because it has been found that plasma cholesterol values tend to run approximately parallel to those of the total fatty acids. He concludes that the plasma cholesterol affords a reliable index not only to the prognosis, but also to the progress of the diabetic patient. The estimation is of particular value when the disease is complicated by infections. In the interpretation of the results consideration must be given to other conditions in which high values are found, such as pregnancy, jaundice and nephrosis.

The Diagnosis of Peptic Ulcer.

T. G. MILLER (*New York State Journal of Medicine*, February 1, 1929) reviews the records of 279 patients suffering from peptic ulcer who were operated upon in the Hospital of the University of Pennsylvania during the last ten years. Peptic ulcer is essentially a disease of men in the middle period of life; 86% of the patients studied were men and 80% of the entire group (men and women) were between thirty and sixty years of age. It was found that 52% of the gastric patients were between forty and sixty years of age. No woman under forty years of age was operated upon for ulcer during the period under review. A characteristic history is one of the most important factors in the diagnosis. The patient in a typical case suffers from prolonged attacks of pain, sometimes of vomiting, nausea and flatulence alternating with periods of freedom from symptoms lasting from weeks to months. Pain, which is by far the most important symptom, was localized in the epigastrium in 90% of the series. A fixed relation to food was noticed in 70% to 80% of the patients. Vomiting was found in three-fourths of the patients, but unless the vomitus consisted of blood or food eaten many hours previously vomiting was not regarded as being of great significance. Periodicity occurred in 60% of the series. The physical examination usually revealed a localized tender spot just above the umbilicus. A mass does not necessarily indicate a malignant lesion, being sometimes due to pyloric spasm and sometimes to an indurated ulcer with adhesions. With regard to the gastric analysis, the patients with duodenal ulcer yielded higher figures for the free hydrochloric acid than those with gastric ulcer, but 59% of the gastric and 31% of the duodenal ulcer patients gave figures that were within or below the accepted normal range. Important evidence of gastric retention, due either to spastic or organic pyloric obstruction, may be given by the test meal. In the series reviewed 94% of the gastric ulcers and 88% of the duodenal ulcers were correctly diagnosed by means of the X rays. The author concludes that the radiological examination is the greatest help in the diagnosis, the history is the next most important factor, while the physical examination and the gastric analysis are only of secondary value.

Blood Transfusion in Severe Sprue Anæmia.

PHILIP MANSON-BAHR (*Journal of Tropical Medicine and Hygiene*, February 15, 1929) records his results from blood transfusion in the treatment of five patients suffering from very severe sprue anæmia and acknowledges the work of Low and Cooke in this connexion. All the patients were desperately ill, one being apparently *in extremis* and another almost in this condition on admission to hospital. One patient was given only seventy cubic centimetres of citrated blood with a brilliantly successful result. From 120 to 550 cubic centimetres were transfused in all other instances. In two instances more than one transfusion was necessary. All the patients recovered rapidly from their anæmia, while the improvement in their mentality and physical vigour was equally striking. Strict dietetic measures were observed as in ordinary sprue and *liquor arsenicalis* was administered, occasionally supplemented by intravenous injections of "Novarsenobillon" (0·1 gramme) every week. The results of blood transfusion appear to be due rather to stimulation of the hæmatopoietic system than mere mechanical replacement of destroyed red blood cells. The striking, immediate and lasting erythroblastic response manifested by these five patients has never been observed in others not subjected to blood transfusion. Manson-Bahr is of the opinion that the transfusion of a larger amount of blood than three hundred cubic centimetres should not be attempted in severe sprue anæmia.

Scarlet Fever.

G. JOUDALEWITSCH, B. LESCHKOW AND O. BAUER (*Klinische Wochenschrift*, January 22, 1929) describe their methods of dealing with scarlet fever and discuss in particular the criteria of cure. The *Streptococcus hæmolyticus* was obtained in the throats of patients, from those with any suppurative complications and also in the air and from the walls of the wards. Only by careful isolation, frequent baths and by transferring patients to a convalescent hospital was it possible to render 70% of the convalescents bacteria-free. Local disinfection, antivirus treatment or the use of serum had little effect on the presence of streptococci. The authors consider that if three normal results are obtained from cultures of the nose, throat and from coughing on blood agar plates and that if there are no septic complications, the convalescent patient can be safely allowed to return home. Skin desquamation alone will not convey the disease to others. The streptococci can remain for long periods (up to ninety days) in the throat and nose. Healthy carriers can be rendered free from organisms within two weeks. Fresh outbreaks are always due to carriers. The authors are opposed to a fixed isolation period of six weeks or until desquamation is completed and consider that bacteriological tests of the respiratory tract are of much more importance. When the patient returns home, the remainder of the family should be tested and any children immunized.

W. STOLTENBERG (*Münchener Medizinische Wochenschrift*, March 1, 1929) summarizes his results in 153 cases of scarlet fever in which serum was administered within the first four days of illness. In the majority of cases (57%) there was definite improvement—the temperature fell, the pulse and appetite improved and the general condition showed much improvement. However, a considerable percentage of patients (43%) either manifested no change or else, if improvement occurred, relapse ensued. These unsatisfactory results bore no relationship either to the degree of infection or to repeated doses of serum. There are no signs whereby an opinion can be given that serum treatment will definitely benefit any given patient. The percentage of patients with late complications was roughly the same in those treated by serum and in those not treated by serum. The author is therefore of the opinion that the scope for the use of the serum is restricted. He would reserve it for primary toxic infections characterized by severe cerebral symptoms, as from his experience further lethal complications are prevented even after a single dose.

Medical Societies.

THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN, BRISBANE.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN, BRISBANE, was held on November 25, 1928, DR. H. MATHEWSON in the chair.

Multiple Herniæ.

DR. K. B. FRASER showed a male baby, aged sixteen weeks, suffering from multiple congenital herniæ. There was an inguinal hernia on each side and a large umbilical hernia. The question arose as to what was the best treatment. The hernial contents were in the habit of coming down and were very difficult to reduce. On one occasion bowel had come down into one inguinal hernia, the baby had been very ill and reduction under general anæsthesia had been necessary. A similar thing had happened on the other side three weeks later. At first the baby had worn an inflated rubber truss; this had been successful for only two weeks. Another doctor had ordered a flexible spring truss.

Congenital Pyloric Stenosis.

Dr. Fraser also showed a baby that had been operated on for congenital pyloric stenosis and was doing very well. This baby and another had been operated on about the same time; the other baby had done well for about six days after operation, then it began to vomit a little and had died suddenly. The cause of death was uncertain, as permission to perform a *post mortem* examination had not been obtained. The baby had been fed on pure condensed milk and had been given two intraperitoneal injections of saline solution after operation.

The baby shown at the meeting was breast fed. It had had to be returned to the theatre a second time, as the peritoneum had separated along the length of the wound and omentum had come through the wound. This baby had a typical history. He had begun to vomit at three weeks, the mother had persisted with breast feeding and had brought him up for advice at eight and a half weeks. Dr. Fraser had found at operation that if there were inclined to be any bleeding, an injection of a few drops of adrenalin at the site of operation was helpful. He also thought that in hospital patients some preoperative treatment was necessary.

Thyreo-glossal Cyst.

DR. C. D. GILLIES showed a young child with a thyreoglossal cyst. The patient when first seen had been pale and ill. There had been a fullness round the throat and on palpation no definite information had been obtained. On examination the mouth had been found clean and the canine teeth had just come through. There had been no rise in temperature. There had been a heaviness in the breathing which developed into stertor when the child was sleeping. When the child was seen one week later, the swelling had gone down considerably and there had been a sausage-shaped swelling below the thyreoid cartilage. The breathing had been more stertorous. The child had then been examined by X rays for the presence of a foreign body, but nothing abnormal had been found. He had then been seen by Dr. S. F. McDonald who thought the general condition was due to the teething and that the stertor was due to the swelling which he regarded as a cyst. The child had been seen one week later and the mother had stated that he had coughed up some phlegm with a little blood and pus in it. After this the difficulty in breathing had disappeared. The submaxillary glands had become tender and five days later had been fluctuant and would probably open in a few days' time. The cyst had gone, there was no fever, but the baby's colour was bad.

Infantile Eczema.

DR. J. W. HEASLOP showed a baby suffering from infantile eczema. The baby also gave a "+++" Wassermann test. Dr. Heaslop said that these conditions were due to incorrect feeding. When the patients were given water these conditions cleared up and then when the patients were given ordinary food for a few days, the condition returned. The baby had been given skim milk and had lost weight; whole milk had then been used and there had been a relapse in the condition.

Dr. Heaslop thought that the eczematous condition was quite independent of the syphilitic condition.

Acidosis.

DR. H. MATHEWSON showed a child, aged ten months, who was suffering from acidosis. The family history was to the effect that three children were alive and two were dead. Apparently they were hæmophiliacs. The child shown had weighed three kilograms (six and a half pounds) at birth and had been breast fed until three weeks previously. There had been regurgitant vomiting for the first four months and the motions had been curdy. At nine months the child had weighed 8·5 kilograms (nineteen pounds). One week previously the mother had weaned the child suddenly and Neaves's food with cow's milk had been given. Two days later the baby had been hot, feverish, constipated, short of breath and panting. Next day it had been restless and choking and the lower extremities livid and mottled.

On admission the bowel had been washed out, saline solution and glucose had been given and oxygen had been administered when he became restless.

DR. J. V. DUHIG then gave an outline of the indications for treatment. This child had been suffering from anoxæmia; the limbs were mottled, there was no vomiting and the condition was one of acidosis. Acidosis was generally not associated with constipation, but with diarrhœa. The child was not getting rid of the carbon dioxide. The respiratory centre was not stimulated and so there was defective oxidation. In order to restore the acid basis there should be sufficient alkalis in the blood to unite with the metabolites. The indication in treatment was to increase the oxygen tension of the alveolar air, to metabolize further the acid products, whatever they were, to supplement the alkali reserve. The child had been put on to alkaline sodium phosphate, two grammes (thirty grains) every two hours.

The prognosis was bad, as there was nothing which would restore the acid base balance.

Tuberculous Adenitis.

DR. S. F. MCDONALD showed a male patient, aged two years and seven months, who was suffering from generalized tuberculous enlargement of all lymphatic glands. Fifteen months previously the glands of the neck had become enlarged and they had been increasing in size since. Facial paresis had appeared a month previously. Numerous glands were palpable in all parts of the body.

A blood count had yielded the following information:

Erythrocytes, per cubic millimetre	2,240,000
Hæmoglobin value	32%
Colour index	0·7
Leucocytes, per cubic millimetre	7,000
Neutrophile cells	81%
Lymphocytes	18%
Eosinophile cells	1%

A gland had been removed from the neck and sections made. These had manifested the presence of tubercle bacilli.

Naval and Military.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 1, 4, 11, 14, 16, 19, 22, 27, 30, 38, 41 and 47 of January 3, 10, 31, February 7, 14, 28, March 7, 21, 28, April 18, 25 and May 16, 1929.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH ROYAL AUSTRALIAN NAVAL RESERVE (SEA-GOING).

Royal Australian Naval Reserve.

Appointments.—Surgeon Lieutenant Donald Dunbar Coutts, M.B., B.S., D.S.O., is appointed sub-District Naval Medical Officer, Williamstown, dated 15th October, 1928.

AUSTRALIAN MILITARY FORCES.

First Military District.

First Cavalry Division.

14th Light Horse Regiment.—The provisional transfer from the Australian Army Medical Corps of Major A. H. Powell, D.S.O., is confirmed.

Australian Army Medical Corps.

Major H. S. McLelland is transferred from the Unattached List and is supernumerary to the establishment pending absorption, 22nd February, 1929.

The provisional appointment of Captain R. E. Douglas is confirmed.

Captain F. W. R. Lukin is transferred to the Australian Army Medical Corps Reserve, 15th March, 1929.

Major E. E. Brown relinquishes the command of the 1st Cavalry Field Ambulance, 18th April, 1929, and is transferred to the Australian Army Medical Corps Reserve, 19th April, 1929.

Australian Army Medical Corps Reserve.

Honorary Captain J. A. Cameron and Honorary Lieutenant A. Wilton are retired, 19th January, 1929, and 15th January, 1929, respectively.

Award of the Colonial Auxiliary Forces Officers' Decoration.

Australian Army Medical Corps.—Major C. J. Weedon.

Second Military District.

Sydney University Regiment.

The provisional transfer to the Australian Army Medical Corps of Major A. B. Lilley is terminated, 31st October, 1928, and he is re-transferred to the Sydney University Regiment and is supernumerary to the establishment pending absorption, 1st November, 1928.

Australian Army Medical Corps.

The provisional appointment of Captain C. C. McKellar is confirmed; the provisional appointment of Captain J. Hughes is terminated, 25th November, 1928, and he is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 26th November, 1928.

Captain T. E. Parker is appointed from the Australian Army Medical Corps Reserve, 15th December, 1928. *To be Captain (provisionally)*—Patrick Gabriel Heffernan, 18th December, 1928. The provisional appointment of Captain A. B. Barry is terminated, 31st December, 1928, and he is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st January, 1929.

To be Majors—Captains H. H. Jamieson and R. A. Money, M.C., 1st January, 1929; Lieutenant N. P. Breden is transferred (provisionally) from the Sydney University Regiment, 1st Division, 18th January, 1929; the regimental seniority of Major W. Vickers, D.S.O., is next after Major A. M. Davidson, O.B.E.; Captain H. G. Leahy is transferred to the Australian Army Medical Corps Reserve, 18th December, 1928.

The provisional appointment of Captain J. Steigrad is confirmed.

Captain M. R. Finlayson is transferred from the Unattached List, 8th February, 1929.

Major C. C. Corlis, M.C., is appointed from the Australian Army Medical Corps Reserve and to be supernumerary to the establishment of Majors, with pay and allowances of Captain, 22nd February, 1929. *To be Captains (provisionally)*—Thomas Whittley Burgess and Archibald Roxburgh Hunt Duggan, 11th March, 1929, and Bruce Thomas Shallard, 20th March, 1929; Captain (provisionally) A. R. H. Duggan is supernumerary to the establishment pending absorption, 11th March, 1929; the provisional appointments of Captains E. M. Sheppard and R. B. Austin are confirmed; Major W. Evans, M.C., is transferred to the Australian Army Medical Corps Reserve, 20th March, 1929.

Australian Army Medical Corps Reserve.

Honorary Captain A. S. Marr is retired, 20th February, 1929.

Colonel E. S. Stokes is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 6th March, 1929; Major G. H. W. Smith is retired, 4th March, 1929.

The resignation of Major G. C. Willcocks, O.B.E., M.C., of his commission is accepted, 31st December, 1928.

Unattached List.

Major T. F. Brown, D.S.O., V.D., is transferred to the Australian Army Medical Corps Reserve, 19th January, 1929.

Third Military District.

Australian Army Medical Corps.

The provisional appointments of Captains J. M. Buchanan and A. J. G. Mackay are terminated, 31st December, 1928. *To be Captains (provisionally) supernumerary to the establishment pending absorption, and to remain seconded*—James Mayo Buchanan and Alan John Grange Mackay, 1st January, 1929; Captain H. G. Furnell ceases to be seconded, 19th December, 1928; Captains W. L. Carrington and J. K. D. Mackenzie, Captain (provisionally) R. W. S. Fox, and Lieutenant E. J. Grieve are brought on the authorized establishment, 1st January, 1929; Lieutenants (provisionally) L. E. Odlum, A. G. Mancy, and E. A. C. Farran are transferred to the Supernumerary List, 1st January, 1929.

To be Lieutenants (provisionally) supernumerary to the establishment pending absorption—John Bastow, 11th February, 1929; Henry Manners Hill, 12th February, 1929, and John Robert Searls, 20th February, 1929.

Honorary Captain F. S. Loughnan is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally) supernumerary to the establishment pending absorption, 4th March, 1929.

The provisional transfer from the Melbourne University Rifles, 4th Division, of Captain H. H. Stewart is confirmed.

The provisional appointment of Captain R. A. Sicree is confirmed; Captain H. G. Mitchell is seconded, 21st February, 1929; the provisional transfer from the Melbourne University Rifles of Captain J. K. D. Mackenzie is terminated, 18th March, 1929, and he is transferred to the Reserve of Officers, 19th March, 1929.

To be Captains (provisionally) supernumerary to the establishment pending absorption—John Gerard Arthur Winter Ashton, 3rd April, 1929.

Captain R. Southby is transferred to the Australian Army Medical Corps Reserve, 1st April, 1929.

Australian Army Medical Corps Reserve.

Honorary Major W. L. Aitken is retired, 26th December, 1928.

To be Honorary Captain—Joseph Ivan Connor, 15th November, 1916 (in lieu of the notification respecting Joseph Joan Connor which appeared in Executive Minute No. 993/1916, promulgated in *Commonwealth Gazette*, No. 176, of 30th November, 1916).

Honorary Captain G. G. Nicholls is retired, 18th January, 1929.

The resignation of Captain F. H. James, M.C., of his commission is accepted, 31st March, 1929.

Reserve of Officers.

To be Captain—Honorary Captain J. I. Connor, from the Australian Army Medical Corps Reserve, 1st January, 1920 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 191/1920, promulgated in *Commonwealth Gazette*, No. 36, of 22nd April, 1920).

Fourth Military District.

Australian Army Medical Corps.

To be Captain (provisionally) supernumerary to the establishment pending absorption—Robert McMahon Glynn, 5th January, 1929.

Fifth Military District.

Australian Army Medical Corps.

The provisional appointment of Captain T. B. Seed is terminated, 31st December, 1928, and he is transferred to the Australian Army Medical Corps Reserve and to be Honorary Captain, 1st January, 1929.

The provisional appointment from the Reserve of Officers of Captain W. S. Cook is terminated, 12th April, 1929. Captain W. S. Cook is re-appointed (provisionally) from the Reserve of Officers, 13th April, 1929. The provisional appointment of Captain H. M. Burns is terminated, 30th April, 1929. Honorary Captain H. M. Burns is re-appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally), 1st May, 1929.

Australian Army Medical Corps Reserve.

Honorary Captains R. H. Hemsted and E. E. Moule are retired, 27th December, 1928, and 1st January, 1929, respectively.

To be Honorary Captain—Alfred Ernest Vivian, 18th February, 1929.

To be Honorary Captain—James Percival Ainslie, 15th April, 1929. Honorary Captain F. M. Wilkinson is retired, 13th April, 1929.

Sixth Military District.

Australian Army Medical Corps.

The provisional appointment of Captain J. S. Reid is terminated, 15th December, 1928.

Captain S. G. Gibson, M.C., is appointed to command the 7th Field Hygiene Section, 9th January, 1929.

To be Lieutenant (provisionally)—George Hugh McQueen, 4th March, 1929.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force—Medical Branch.

Appointment.—Frederic Hobart James, M.C., to a commission with the rank of Flight Lieutenant, 1st April, 1929.—(Ex. Mins. Nos. 84 and 85.)

Correspondence.

DAMAGES IN MEDICO-LEGAL CASES.

SIR: There exist certain principles that should underlie the assessment of damages due from one person to another in cases of accident, injury and misadventure. This is well recognized in workers' compensation acts, insurance schemes *et cetera*, but it seems to be ignored in cases of professional accident (or malpractice).

It is the duty of the medical profession with the aid of its legal advisers to enunciate these principles and to see that juries are instructed in them by the judge; for once a jury comprehended them, we would see the end of excessive, not to say vindictive or sentimental, verdicts. Failure of the judge in this duty should afford sufficient reason for appeal against the amount of damages on the ground of misdirection by the judge.

Consider: 1. The previous and probable future capacity of both parties to the complaint and the effect of the malpractice on these. (It is imbecile to award damages that cannot be collected or in being collected ruin the defendant.) Income tax assessments for a series of years should give evidence of the financial position (and the financial injury) of both parties.

2. Reimbursement for actual suffering, expenses, losses, worry *et cetera* up to the date of the action and thereafter, if necessary.

3. Standard rates of compensation apply in industry, for example, rates for each limb, each organ, the totality of injury, the duration of partial or complete disablement; this should apply in professional accident also. Economists and governmental authorities appear to assess the economic value of a life at £2,000; that is the limit of liability on a public railway for death arising from traffic accidents. Could there also be a standard economic cost of living for the assessment of damages for partial or total disablement?

4. Wisdom is easy after the event, especially to those with no real knowledge of the difficulties of practice. An unfortunate concatenation of circumstances, weariness, errors of judgement, ignorance (no one can know everything) are always liable to occur and should not provide occasion for crippling crimes. Heavy penalties will prevent the profession attempting the difficult or the uncertain.

5. The judge, assisted by the counsels' previous efforts, should explain to the jury the effects on both parties, so that the jury can exercise a well-informed judgement, instead of being left to act on assumptions "that doctors are made of money" and "that doctors make their money easy" (remarks I have frequently heard) and penalizing the defendant as if he were concerned in a criminal instead of a civil action.

The British Medical Association should institute a compulsory insurance of all its members against damages for malpractice and so spread the risk over the whole profession.

Yours, etc.,

MARY C. DE GARIS.

Geelong,
May 5, 1929.

THE TAKATA-ARA REACTION OF THE CEREBRO-SPINAL FLUID.

SIR: In 1928 Dr. Takata and Dr. Ara published in Tokyo a paper entitled: "A New Colloid-Chemical Reaction of the Cerebro-spinal Fluid and the Results Obtained in its Application" (*Ueber eine neue Kolloidchemische Liquorreaktion und ihre praktische Ergebnisse*). The test is one of those which to the laboratory man has many charms on account of its neatness and the facilities which it offers. In order that results with the test may be made known, I would suggest that the test be adopted in our public hospitals.

The test is as follows:

Reagents required: (i) A 10% solution of sodium carbonate; (ii) a 0·5% solution of perchloride of mercury; (iii) a 0·02% solution of fuchsin. Proceed as follows: Take one cubic centimetre of clear cerebro-spinal fluid, add one drop (0·033 cubic centimetre) of the sodium solution, then add 0·3 cubic centimetre of a mixture of (ii) and (iii) in equal parts. The result is read off at once, in a quarter of an hour in half an hour, in twelve to twenty-four hours, according to the resulting change. A diagnosis is made of metalues if a blue-violet flocculent precipitate appears. The diagnosis of meningitis is made if no precipitate appears and the liquid changes colour to pink.

Yours, etc.,

A. E. FINCKH.

The Sydney Clinical Research Laboratories,
227, Macquarie Street, Sydney.
May 15, 1929.

CEREBRAL ABSCESS.

SIR: May I make some corrections on case reported in the journal of April 27, 1929, as being shown by me at a meeting of the Queensland Branch on October 11, 1928.

When first seen the history was of a left acute *otitis media* of no more than four days' duration, hence the "simple" mastoid operation. At this operation the internal jugular vein was partially resected, the common facial vein ligated. The lateral sinus was surrounded by pus and the

contents of the sinus consisted of pus. These conditions naturally aroused doubt as to the previous history and the patient's mother, when subsequently questioned, admitted that the ear had given trouble on and off for two years. Four days later the rapid onset of violent nystagmus and vomiting, accompanied by double papillædema and the equally rapid deterioration of the patient's general condition with some retention of hearing, was regarded as suggestive of cerebellar abscess, considering also the extensive perisinus abscess and the state of the lateral sinus itself.

At the second operation the cerebellum was explored thoroughly with negative result, a little pus was then seen to be escaping in the temporal region and the *dura* was elevated until at the region of the apex of the petrous bone there was a large escape of pus, followed by a very free flow of cerebro-spinal fluid.

A soft rubber tube drain was inserted.

The patient escaped a general meningitis. About ten days later a facial paresis made its appearance and for about a week gradually got worse and the neurologist after careful examination diagnosed a cerebellar abscess. However, no further operative treatment was carried out, the paralysis gradually disappeared and the wound gradually healed.

The tube was extruded from the wound. The patient is well today, except for a little evidence of some necrosis in the attic region and there is now nine months after the first operation a slowly developing spastic condition of the left facial nerve.

Two points to be stressed are: (i) the original faulty diagnosis of an acute condition, instead of a chronic, leading to incompleteness of the mastoid operation; (ii) at no time was there complaint of headache or pain, except slight earache, in fact the patient complained of no other symptoms and when desperately ill was in good spirits, in spite of the undoubted increased intracranial pressure.

Yours, etc.,

ERNEST CULPIN.

Wickham Terrace, Brisbane.
May 4, 1929.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following information has been communicated to us by the Executive Committee of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929.

SECTIONAL ARRANGEMENTS.

Section of Medicine.

The provisional programme drawn up by the officers of the Section of Medicine is given below.

"Peptic Ulcer and the Conditions which Simulate it," by Dr. C. T. de Crespigny, of Adelaide. Dr. J. R. Bell, of Melbourne, and Dr. S. F. McDonald, of Brisbane, will also speak.

"Œdema," by Sir Richard Stawell, of Melbourne. Professor R. Camalt Jones, of Dunedin, will be the second speaker.

"Cardiac Arrhythmias," by Dr. Alfred Webster, of Perth. This paper will be illustrated by lantern slides. Contributions to the discussion are invited.

"Protein and Vaccine Therapy." The inclusion of this subject in the programme will depend on the offer of papers within the next fortnight. The response hitherto has been disappointing. Intending readers of papers are asked to communicate with the Honorary Secretary of the Section, Dr. Cotter Harvey, 235, Macquarie Street, Sydney. It is proposed to complete the programme at an early date.

Section of Naval, Military and Air Force Medicine and Surgery.

It has been decided that the question of first aid and invalid transport will be discussed at one of the meetings of the Section of Naval, Military and Air Force Medicine and Surgery. A paper on the subject will be read by Dr. H. R. G. Poate, of Sydney.

Section of Pædiatrics.

The following programme has been arranged for the Section of Pædiatrics.

Tuesday, September 3, 1929.

2 p.m.—President's address, Dr. S. W. Ferguson.
"Asthma and Allergic Conditions in Childhood," by Dr. C. Sutherland, of Melbourne.
"The Electrocardiogram in Childhood," by Dr. H. L. Stokes, of Melbourne.

Wednesday, September 4, 1929.

9.30 a.m.—"Human Serum in Poliomyelitis," by Dr. Jean Macnamara, of Melbourne.
11 a.m.—Subject not yet determined.
2 p.m.—Clinical meeting at the Royal Alexandra Hospital for Children, Camperdown.

Thursday, September 5, 1929.

9.30 a.m.—"Infant Feeding to Six Months," by Dr. H. Boyd Graham, of Melbourne.
"Restoration of Breast Milk Feeding," by Dr. Guy Springthorpe, of Melbourne.
Visit to Tresillian Mothercraft Training School, Petersham.
2 p.m.—"Bone Dystrophies," by Dr. R. B. Wade, of Sydney (in conjunction with the Section of Orthopædics and the Section of Radiology and Medical Electricity).

Friday, September 6, 1929.

9.30 a.m.—"Chronic Pulmonary Diseases Secondary to Diseases in the Upper Air Passages," by Dr. J. B. Douglas Galbraith, of Brunswick (in conjunction with the Section of Medicine, the Section of Oto-Rhino-Laryngology and the Section of Radiology and Medical Electricity).
11 a.m.—"Natal and Neo-natal Mortality and Morbidity," by Dr. P. L. Hipsley, of Sydney (in conjunction with the Section of Obstetrics and Gynæcology and the Section of Preventive Medicine and Tropical Hygiene).
2 p.m.—Clinical meeting at the Royal Alexandra Hospital for Children, Camperdown.

Saturday, September 7, 1929.

The programme has not yet been arranged.

Dr. M. J. Plomley, 233, Macquarie Street, Sydney, the Honorary Secretary of the Section, will be glad to receive the names of members who are prepared to take part in the discussions on the subjects selected.

Dr. R. H. Crisp, of Perth, has accepted the position of Vice-President of the Section of Pædiatrics.

Section of Oto-Rhino-Laryngology.

Dr. J. Stoddart Barr, of Hobart, and Dr. H. J. Gray, of Perth, have accepted the position of Vice-Presidents of the Section of Oto-Rhino-Laryngology.

Excursions.

The following excursions will be available to members of Congress. The list has been arranged by the Excursions Committee in conjunction with the New South Wales Government Tourist Bureau. Further information will be published at a later date.

Half Day Excursions.

To Palm Beach, to Taronga Park, to the North Shore Bridge (September 7), to the National Park, Harbour trip, to Bobbin Head (Kuring-gai Chase), to Galston Gorge, Pennant Hills, Baulkham Hills School, to Vaucluse, South

Head, Botany, La Perouse, to Lady Carrington Drive, to Hornsby, to French's Forest and Elanora golf links, to Hawkesbury and Berowra, through the city, gardens, Vaucluse House, Coogee, Centennial Park, to Newport, Bayview, Bilgola, to the National Park and Cronulla by water, to Windsor and Cattai Creek.

Visits to factories and public buildings, to the Australian Museum and to the Art Gallery will be arranged as required.

Whole Day Excursions.

To Bulli and Cataract Dam, to Kurrajong Heights, to Brooklyn and Wiseman's Ferry, to the Blue Mountains, to Avon Dam, to the steel works, Newcastle, to Burragorang and Mulgoa, to Port Kembla and Lake Illawarra.

Excursions to More Distant Places.

Jenolan Caves, to Kangaroo Valley and to Canberra.

Membership.

A notice has recently been circulated among the members of the several Branches of the British Medical Association in Australia and New Zealand giving information concerning Congress and containing a form of application for membership of Congress. If any member fails to receive his copy in the ordinary course of post, he should notify the joint Honorary Secretaries of Congress, so that a copy may be sent to him.

Books Received.

PERCUSSION OF THE CHEST, by J. B. McDougall, M.D. (Glasgow), F.R.C.P. (Edinburgh), F.R.F.P.S. (Glasgow); 1929. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 151, with illustrations. Price: 6s. net.

GONOCOCCAL URETHRITIS IN THE MALE: FOR PRACTITIONERS, by P. S. Pelouze, M.D.; 1929. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 357, with illustrations. Price: 25s. net.

ALCOHOL AND HUMAN LIFE, by Courtenay C. Weeks, M.R.C.S., L.R.C.P., with a Foreword by Sir Thomas Barlow, Bart, F.R.C.P., M.D., F.R.S.; 1929. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 213. Price: 3s. 6d. net.

Diary for the Month.

JUNE 25.—New South Wales Branch, B.M.A.: Council (Quarterly).
JUNE 26.—Victorian Branch, B.M.A.: Council.
JUNE 27.—New South Wales Branch, B.M.A.: Branch.
JUNE 27.—South Australian Branch, B.M.A.: Branch.
JUNE 28.—Queensland Branch, B.M.A.: Council.
JULY 2.—Tasmanian Branch, B.M.A.: Council.
JULY 2.—Eye, Ear, Nose and Throat Section, South Australian Branch, B.M.A.
JULY 3.—Victorian Branch, B.M.A.: Branch.
JULY 3.—Western Australian Branch, B.M.A.: Council.
JULY 4.—South Australian Branch, B.M.A.: Council.
JULY 4.—Section of Orthopædics, New South Wales Branch, B.M.A.
JULY 5.—Queensland Branch, B.M.A.: Branch.
JULY 9.—Tasmanian Branch, B.M.A.: Branch.
JULY 9.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 10.—Central Northern Medical Association, New South Wales.
JULY 11.—Victorian Branch, B.M.A.: Council.
JULY 11.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 12.—Queensland Branch, B.M.A.: Council.
JULY 16.—Tasmanian Branch, B.M.A.: Council.
JULY 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

MACKAY HOSPITALS BOARD: Resident Medical Officer.
REPATRIATION COMMISSION: Junior Resident Medical Officer.
ST. MARGARET'S HOSPITAL, SYDNEY: Resident House Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Toowoomba Friendly Societies Medical Institute. Mareeba Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

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All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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